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IMMUNIZATION AGAINST ANTHRAX

No more serious problem has confronted the veterinary profession, particularly during recent years and in certain parts of the United States, than the control of anthrax. The disease is generally regarded as being the limiting factor in the up-building of a productive animal industry in those sections of the United States which have been frequently referred to as notorious anthrax districts.

The vaccine developed by Pasteur was for many years the only agent at the disposal of veterinarians for any system of prophylactic treatment against the disease. It appeared to be highly successful in the hands of some veterinarians, during some years, and in some localities. In the hands of other veternarians, or in certain years, or in certain districts, failure attended the use of the vaccine and various excuses were given for these failures.

A few years ago, new hope was held forth and a solution for the problem appeared to be at hand in the form of the so-called simultaneous method of vaccination with serum and vaccine. In many distructs where the disease never took on a particularly virulent form, the results attending vaccination by the simultaneous method were uniformly good. On the other hand, in

the so-called notorious anthrax districts, vaccinated animals died of anthrax just as before. Any form of vaccination apparently failed to produce a degree of immunity sufficient to protect the animals against the highly virulent strains of the anthrax organism which were indigenous to these localities.

The next improvement was in the form of a stronger, less-attenuated vaccine, designed to develop a higher degree of immunity in the vaccinated animals. Advantage was taken of the knowledge gained through the hyperimmunization of animals used to produce anti-anthrax serum, and the same process, to a limited degree and on a smaller scale, was applied to animals in the field.

It is generally conceded that the degree of immunity produced by vaccination will be in direct ratio to the extent of the reaction produced by the vaccine. The practical application of the stronger vaccine, however, has not worked out satisfactorily. As a matter of fact, some of the reactions which have followed the use of these vaccines have been so severe as to render the animals unfit for work for variable periods following vaccination. Unfortunately, the times which appear to be the most desirable for vaccinating animals are likely to be either when the animals are not in the best physical condition to withstand severe reactions, or when they are badly needed for farm operations. The inability of owners to work the vaccinated animals, for variable periods following vaccination, has seriously interfered with the spring work, so much so that many owners have expressed the opinion that they would rather take the chance of losing the animals from anthrax than have them incapacitated for work at such an important time of the year.

With the knowledge of these facts and with a desire to develop an immunizing agent that would at the same time protect the animals against anthrax and make it possible for them to continue at work, Dr. C. E. Salsbery took up the problem of developing anthrax aggressin. The results of some of his studies are reported in a paper appearing in this issue of the JOURNAL. We feel that a great deal of credit should be given Dr. Salsbery for his patience and perseverance in overcoming the many obstacles which beset his path. Anthrax aggressin appears to have most of the advantages of the other immunizing agents against anthrax and at the same time some advantages not possessed by them.

The question arises in our mind whether or not the immunity induced by vaccines is not the result of aggressin which is pro-

duced locally at the site of the injection. If such be the case, what will be the difference, if any, in the immunological mechanism between the case where the aggressin is produced locally by the tissues of the vaccinated animal, on the one hand, and the case of an aggressin-treated animal, on the other, where the aggressin is merely transferred from animal to animal?

Unfortunately for the new product, but at the same time fortunately for the owners of live stock in the anthrax districts, the disease was not very serious during the year 1925, when anthrax aggressin first became available for practical field trials to determine its ability to protect animals against the disease. We understand that these field trials are being continued, just as frequently as opportunities are offered, and it is certainly to be hoped that anthrax aggressin will gain a place in the same rank with the other aggressins which have recently been added to our list of immunizing agents, namely, those for blackleg and for hemorrhagic septicemia.

TWO IMPORTANT OPINIONS

Attorney General N. T. Gentry, of Missouri, recently rendered an opinion containing a ruling relative to compensation for the vaccination of hogs. The opinion was substantially as follows:

No one is authorized to administer serum to hogs, with or without virus, except in case of emergency, when it may be administered by anyone, but it must be done without charge, and for any person to make a charge, unless a regularly registered veterinarian, would be a violation of the Veterinary Practice Act of Missouri.

In another opinion, a few days later, made by Attorney General Carlstrom, of Illinois, to Dr. F. A. Laird, state veterinarian, it is held that farm advisers may vaccinate hogs as an accommodation but not for compensation. It is also held that a farmer may not vaccinate the hogs belonging to his neighbor in exchange for work. The opinion says in part:

"The question in the case of a farm adviser depends largely upon the terms of his contract with the farmers who have hired him.

"If vaccinating the various farmers' hogs is one of his duties or is an incident to his duties, then I am of the opinion that he is receiving compensation directly for so doing and will come under the law regulating veterinarians. It would likewise seem that,

if he vaccinated a farmer's hogs as a demonstration as part of instruction to the act of vaccination, he would come within the provisions of the act.

"If, however, he performs the act merely as a matter of accommodation, then, he would not be receiving compensation.

"If a farmer, for the works his neighbor has performed, recompenses his neighbor by vaccinating his neighbor's hogs, he would come under the act. It cannot be assumed, however, that it was the legislative intent to say that a farmer may not vaccinate his own hogs."

EXECUTIVE BOARD ELECTION

Ballots asking for nominations of candidates for member of the Executive Board to represent District No. 6 were mailed to all paid-up members in the district, February 17, 1926, exactly six months prior to the opening date of the annual meeting to be held in Lexington.

Five days later, twenty-two ballots have been returned, signed by members located in five different states, nominating thirteen candidates, representing six different states in the district. If you are located in District No. 6 and failed to receive a ballot, drop a line to the Secretary and ask him why.

APPLICATIONS FOR MEMBERSHIP

(See October, 1925, JOURNAL)

FIRST LISTING

BATTEN, F. B — 179 Park Ave., Lexington, Ky.
D. V. S., Ohio Veterinary College, 1894
Vouchers: W. F. Biles and W. W. Dimock.

BIRDWHISTELL, ROBERT N. — 1047 Oak St., Columbus, Ohio
D. V. M., Cincinnati Veterinary College, 1914
Vouchers: E. P. Maxwell and F. A. Lambert.

BONIFIELD, H. M. — Warren, Ind.
D. V. M., Ohio State University, 1924
Vouchers: J. H. Mills and W. B. Craig.

ELIASON, C. O. — Benson, Minn.
V. S., Ontario Veterinary College, 1907
Vouchers: A. J. Thompson and M. H. Reynolds.

HANNA, A. F — Fortuna, Calif.
D. V. M., San Francisco Veterinary College, 1912
Vouchers: John L. Tyler and C. M. Haring.

HOLMES, CHARLES HAROLD — 144 E. Short St., Lexington, Ky.
V. S., B. V. Sc., Ontario Veterinary College, 1924
Vouchers: Chas. E. Hagyard and W. W. Dimock.

JOHNSTON, ERNEST F. — Carp, Ont.
B. V. S., Ontario Veterinary College, 1922
Vouchers: C. D. McGilvray and R. A. McIntosh.

McBROOM, GEORGE W. — Atwater, Minn.
D. V. M., Chicago Veterinary College, 1912
Vouchers: H. C. H. Kernkamp and A. J. Thompson.

SNORTUM, ERNEST P — Canby, Minn.
D. V. M., McKillip Veterinary College, 1917
Vouchers: A. J. Thompson and H. C. H. Kernkamp.

VANSANT, JOSEPH W. — Huntingdon Pike and Shady Lane, Fox Chase, Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1902
Vouchers: G. A. Dick and C. J. Marshall.

Applications Pending

SECOND LISTING

Allerton, Fred R., Hamlin, Kans.
Alston, Joseph Thomas, Tupelo, Miss.
Banister, Robert Martin, Sumner, Miss.
Barger, Edgar H., University Farm, Davis, Calif.
Blaser, Charles D., 711 Genesee St., Buffalo, N. Y.
Crawford, William R., College Ave., College Park, Md.
Emmerson, M. A., Iowa State College, Ames, Iowa.
Gates, Charles H., Liberty, Ind.
Hagyard, Edward W., 144 E. Short St., Lexington, Ky.
Killpack, M. Lloyd, R. F. D. No. 3, Box 183, Sandy, Utah.
Kinney, James Roser, Netcong, N. J.
Kreidler, Henry E., Spartansburg, Pa.
McIntosh, G. E., 309 Central Ave., Louisville, Ky.
McPhail, James A., Manistique, Mich.
Newman, W. P., Perham, Minn.
Sarde, Robert M., Camden, Del.
Sebastian, George Fink, Rehrersburg, Pa.
Snyder, Orrin W., Liberty, Ind.
Smith, Fraser A., 144 E. Short St., Lexington, Ky.
Stewart, Robert Hyram, Belzoni, Miss.
Waghorn, T. G., Brockville, Ont.
Winkler, J. A., Prospect & Elm Sts., Newport, Ky.

Reinstated

Bateman, W. C., 495 18th St., San Bernardino, Calif.
Burns, A. H., Box 91, Bucklin, Kans.
Gates, W. L., Box 417, Clarksdale, Miss.
Gordon, W. D., Morristown, Ind.
Rhea, R. L., 307 Jones Ave., San Antonio, Tex.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Academy of Medicine, 17 W. 43rd St., New York, N. Y. Mar. 3, 1926. Dr. C. G. Rohrer, Secretary, 40 W. 61st St., New York, N. Y.

Keystone Veterinary Medical Association. Philadelphia, Pa. Mar. 24, 1926. Dr. G. A. Dick, Secretary, 39th St. & Woodland Ave., Philadelphia, Pa.

Ohio State University Conference of Veterinarians. Columbus, Ohio. Mar. 24-25-26, 1926. Dr. O. V. Brumley, Acting Dean, Ohio State University, Columbus, Ohio.

Massachusetts Veterinary Association. American House, Boston, Mass. Mar. 31, 1926. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

DISEASES AFFECTING THE CENTRAL NERVOUS SYSTEM OF THE CANINE AND FELINE*

By O. V. BRUMLEY, *Columbus, Ohio*

College of Veterinary Medicine, Ohio State University

The diagnosis, if it can be made, is the important factor or keynote in efficiently treating and handling diseases of the central nervous system of our small patients. I think it is generally recognized that this important part of the animal's anatomy represents a very complicated mechanism and therefore needs very careful consideration from both a physiological and pathological standpoint. The practitioner is naturally confronted, therefore, with diseases that produce symptoms not clear-cut as we find them in many other conditions in these animals.

Careful observation and experience, with a study of the functions of the central nervous system, will produce results in arriving at definite conclusions and the degree of success in this regard will depend to quite an extent upon how careful we are in studying all of our cases as they are presented to us. I am referring in this connection not only to the symptoms the animal shows at the time of the first examination, but to the fact that the progress of all conditions should be studiously observed in contrast to the symptoms first presented and, whenever possible, in case of death, autopsies should be held, in order to connect the symptoms with the portion of the central nervous system affected.

Autopsies in many cases do not reveal sufficient changes to account for the prominent symptoms shown during life and to have caused the death of the animal. In these cases, when possible, microscopic examinations of different portions of the brain and membranes should be made. It is only after complete examinations of these patients that we can expect to be reasonably expert in the diagnosis of these diseases.

In the determination of diseases of the nervous system, natural habits of these animals must be taken into consideration. How many times have we observed our patients show nervousness as a result of being in strange hands or environment. Shyness in some cases or tendency for viciousness in others may be due entirely to conditions surrounding the individual animal. Therefore, as a suggestion, I would recommend that in your anamnesis

*Presented at the sixty-second annual meeting of the American Veterinary Medical Association, Portland, Oregon, July 21-24, 1925.

you make definite inquiry concerning the habits of the animal. The master of these small patients will, in the majority of cases, be able to give you facts along this line which will be of inestimable value in arriving at a positive conclusion. Too much emphasis cannot be placed on the fact that kindness and careful handling and manipulation of the patient is always in order. Therefore, it is actually necessary, in many cases of nervous conditions, to make the distinction between habits, etc., and pathological or functional disturbances of the nervous system.

Just for a few moments let us consider what functions of the central nervous system we should consider from a practical standpoint.

The cortex: The cortex of the cerebral hemisphere is the seat of all psychic function, such as thought, the will, and sensation, and all efferent nerve fibers originate here. The voluntary motor fibers also originate in the cortex, pass through the pons to the medulla oblongata, where they cross to the opposite side and communicate with the motor nerves of the extremities. All sensory nerve fibers and fibers of special senses, which conduct perceptible impulses to the brain, terminate in the cortex. The cortex of the cerebrum, then, being the seat of the mind and of volitional mobility, it follows that any destructive process affecting this portion of the brain will produce psychic or mental disturbances as well as impaired mobility and sensation on the opposite side of the body, the degree of impairment depending upon the extent of the lesion.

The midbrain, crura cerebri, corpora quadrigemina, and optic thalami: This portion of the brain is the seat of harmony of motion and equilibrium. As some of the cranial nerves arise here, the fifth pair being the most important of these, any disease, destructive process or undue pressure operating on this part of the brain will produce sensory and motor disturbances in the region of the face, lips, eyes, ears, and part of the tongue, and, if extensive, the entire organism may be affected. Involuntary movements of the limbs, head, neck and eyes are the most common symptoms resulting.

The cerebellum: The functions of the cerebellum or hindbrain are not fully known, but it is regarded as being closely connected with locomotion and equilibrium. It is also thought to be the seat of the muscle sense and assists in the coordination of the muscles of the same side of the body and, if either half be injured or diseased, the animal will exhibit muscular and motor

disturbances of the same side, varying in degree from slight muscular incoordination to spasmodic movements, or it may walk in a circle or crowd or roll toward the injured hemisphere.

The brain, because of its position, protected by the bones of the cranium, cannot be examined directly. Diseases of this organ can be recognized only by observing the disturbed functions produced after pathological changes have occurred. It is necessary, therefore, to examine carefully the functions of the brain before one can arrive at definite conclusions.

As the brain is the seat of the mind (thought), feeling, consciousness, sensibility, and volitional mobility, any disturbances or impairment of these functions must be attributed to some pathological change in that organ.

Taking up the examination of the brain by examining its functions in the order named above, the psychic function or mental condition will be considered first.

Psychic Disturbances

Any variation from the normal mental condition is manifested by abnormal excitability, or abnormal depression.

Mental excitement: Abnormal mental excitement is caused by cerebral irritation involving the cortex particularly. This may be due to hyperemia, inflammatory changes, excessive heat or toxic influence. The degree of excitement may vary from restlessness to mania. In these attacks docile animals may become vicious, bite animate or inanimate objects, or even bite their own flesh; may stand up on their hind legs, froth at the mouth, and the eyes show a vacant, staring expression with the conjunctiva injected. These symptoms may gradually subside of they may terminate in spasms and convulsions.

Mental depression: This may be defined as a dulling of the psychic functions and may vary in degrees from dullness to coma. Mental depression is shown by the animal taking less interest in its surroundings than usual, drooping of the head and tail, refusing to obey commands or obeying slowly or clumsily; it assumes somnolent or lethargic attitudes or may wander aimlessly about, running into objects, etc. These are the milder manifestations of depression and are seen in subacute and chronic diseases, as the early stages of distemper and rabies, in severe fibrile diseases, and in icterus and uremia. Other degrees of mental depression are shown by somnolency, a condition in which the animal appears to be asleep, but from which it may be

roused; or sopor, deep sleep from which it is difficult to rouse the animal, and coma, or complete unconsciousness. These conditions are produced by more severe or extensive lesions. They are seen in compression of the brain, cerebral hemorrhage and tumors of the brain. They may be accompanied by motor disturbances in addition to the mental symptoms, since these conditions may involve the deeper structures of the brain as well as the cortex.

Sensibility: Disturbance of sensation may be considered as of two types: (a) pathological excitation or hyperesthesia and (b) pathological depression or anesthesia.

Hyperesthesia, when observed in the small animals, is usually due to some of the infectious diseases, and is seen in the early stages of rabies, in tetanus, and in some of the milder diseases of the cerebrum, as hyperemia and acute cerebritis. Hyperesthesia is manifested by abnormal movements of the animal, which are entirely out of proportion to the stimulus applied. For example, slamming the door or clapping the hands may so excite the animal that it will fall to the floor or ground in spasms. Local or peripheral hyperesthesia is of little or no importance in small animal practice.

Anesthesia: This is a condition in which there is a complete loss of sensation. Hyperesthesia indicates a condition in which tactile sensibility is merely decreased. Diminished sensibility may be general or complete, affecting the entire animal, as in subacute or chronic inflammatory conditions of the cortex of the brain and its coverings. It may be partial or incomplete, affecting one entire side, having its origin in one hemisphere, that of the opposite side, or it may be local, or circumscribed, when more or less extensive areas of the cerebrum are involved. Depression of sensibility is determined by applying some stimulus, which, when applied to the normal animal, will cause pain. To test sensibility, the skin is pricked with a needle or pin, pinched or burned with a heated instrument. If the animal fails to react, that is, does not show pain by crying out, whining, or trying to get away from the irritant, the area or part tested is anesthetic.

Motility: Disturbances of motility arising from the brain vary in degree from slight incoordination to complete paralysis. They will vary in extent and character depending upon the location and size of the lesion. Disturbed motility may be classified into: (1) hyperkinesis, or exaggerated action, as seen

in spasms and involuntary movements; and (2) akinesis, or decreased action, such as occurs in loss of the musuclar sense and in paralysis. Disordered motility may arise from pathological changes in the brain or in the spinal cord. In examining disturbances of motility, it is sometimes impossible to locate definitely the seat of the lesion. However, if the impaired function is accompanied by mental disturbances, the brain may be regarded as being the seat of the lesion. If, on the other hand, no mental disturbances are noted, it is assumed that the lesion is in the cord. Further, the portion of the animal affected will often point to the origin of the disturbance. Hemiplegia, or paralysis of one-half of the body, and monoplegia, paralysis of a single organ or part, indicate that the disturbance is of cerebral origin, while paraplegia, a paralysis of a portion of both sides, indicates spinal paralysis.

The following are the most important diseases affecting the central nervous system. It is not our intention to describe any of these diseases in detail, as it would consume entirely too much time, and, therefore, I will confine my statements, as far as possible, to the practical application. In some instances certain diseases have as their early manifestations symptoms indicating a primary involvement of the central nervous system, while in other individual cases the disease may attack some one of the other systems of the body. This is true in certain forms of distemper, rabies, and a few others. Therefore, it will be necessary to discuss briefly such diseases as affect the central nervous system even though the primary lesion is not always found in this location.

Hyperemia and Congestion

Hyperemia and congestion of the brain is not so frequently observed, but is of sufficient importance to be mentioned briefly in this particular discussion. We recognize two particular forms of this condition, the active and the passive congestion, both of which are observed. In active hyperemia or congestion it is generally brought about by violent exercise, excitement, blows and concussions over the head, or as it does occur, in some instances, due to excessive heat. In young puppies sometimes it occurs during the eruptive stage of the development of the permanent teeth.

As a secondary consideration, we must keep in mind the possibility of this condition developing as a result of other diseases

or, as is frequently the case, associated with other diseases. Here must be mentioned particularly rabies and the nervous form of distemper. The passive congestion is considered mainly mechanical and is produced by any mechanism which impedes the flow of blood from the brain. Here might be mentioned tight collars or pulling on the leash. These bring about this condition by the compression of the jugular vein. Tumors and enlarged thyroid glands (goiter), sometimes valvular insufficiencies of the heart and chronic pneumonia may also produce it. The principal manifestations of this condition are those of restlessness, shown by the animal changing its position or wandering about in an aimless fashion, excitement, irritability, with a tendency to bite shown by some animals, although, unless produced by rabies, the animal does not show any aggressive spirit. In the most severe type there will be observed spasms and convulsions.

If we look at the conjunctiva and the mucous membrane of the mouth, they are usually found to be injected, the pupil of the eye is contracted, the animal shows a rather vacant and staring expression. The head shows a feeling of warmth which is abnormal, the appetite is practically gone and vomiting occurs in some cases. It is rather significant that these symptoms develop suddenly and in the active condition usually disappear in the course of a few hours or two or three days. In the passive congestion of the brain, the symptoms are mainly those of depression.

The treatment of these cases should be largely to keep the patient in a cool, quiet, rather dark place, away from all noise and excitement. Do not attempt to force the animal to eat, as it will simply aggravate the symptoms. Plenty of fresh milk and tempting bits of meat should be offered frequently. In order to divert the blood from the brain in these cases, a laxative is indicated. Small doses of pilocarpin may be employed in order to relieve the congestion. When the excitement is severe in dogs, we recommend the usual administration of morphin in order to control the symptom; in cats ether perhaps would be more applicable to check the excited condition temporarily. In cases where depression is permanent, as in passive congestion, stimulants would naturally be recommended. Oil of camphor, small doses of caffein or atropin sulphate, as subcutaneous injections, have given good results in many cases. After convalescence has been established, give the animal plenty of nourishing food, laxative in character.

Encephalitis

This condition consists in an acute inflammation involving the brain and its covering. In practice it is very difficult to make any clear distinction between this condition and meningitis. Therefore, we usually classify the condition as meningo-encephalitis. This condition is caused in the majority of instances from thermic conditions, or some infectious disease, such as rabies or distemper. In some cases in dogs we have observed the condition developing as a result of parasitic invasion of the auditory canal. This perhaps is more often observed in foxes and rabbits than in dogs. The principal factors observed in this case are very marked psychic or motor disturbances, or both, and rather extreme restlessness, in which the animal may howl or bark continuously. They will run about aimlessly, often running against objects, showing difficulty in seeing.

Spasms and convulsions followed by unconsciousness are observed frequently. In the severe cases animals may die during the period of one of these convulsions. This condition is mentioned inasmuch as some of the symptoms shown by these animals are quite similar to that found in rabies and the nervous form of distemper. Therefore, there should be extreme caution used in handling any of these small animals showing such manifestations. We have found it rather difficult to control these symptoms by treatment, especially when the condition has become severe. It would seem necessary therefore, to apply more or less symptomatic treatment in the way of narcotics and stimulants if they are needed. In these more severe forms, we have found treatment is of little avail.

Apoplexy or cerebral hemorrhage is not very frequently observed in small animals. We have seen a few cases during the past few years, most of which were in old dogs. Upon investigation of these cases, it was found that there had been degenerative changes in the structure of the arteries which, upon exercise, ruptured and produced death in a few minutes. In some old animals that have been affected with rheumatism, there result disturbances of the circulation and upon rather severe exercise there is very apt to be a hemorrhage.

The principal symptoms observed in these cases are those of paralysis, which comes on very suddenly after excitement or exercise. The animal drops down suddenly and is convulsed with muscular spasms. The animal may die in this particular

attack or the attack may pass away, the animal losing consciousness for a while. Respiration becomes slow and labored. In these cases where death does not follow at once, it usually leaves a partial or complete paralysis, monoplegic or hemiplegic in character, depending on the size and location of the hemorrhage. Animals developing these symptoms suddenly should be placed in a moderately cool, well ventilated, quiet room, away from all noise and excitement. Cold applications should be used around the head and around the region of the neck. Where the excitement is extreme, sedatives should be administered at once. Where the animal makes a partial recovery, small doses of potassium iodid should be used, together with an active laxative, the object being to assist in the absorption and elimination of the results of the hemorrhage. Following this treatment, when paralysis is present, the electric battery and strychnin in small doses given daily should be used.

Paralysis Due to Various Causes

The special nerves emanating from the central nervous system should receive careful consideration by the practitioner. It often happens that nerves supplying the vital functions are either impaired or paralyzed, which causes serious difficulty to our patient. Facial paralysis, paralysis of the tongue and some of the other organs should be given consideration. It is generally believed that these forms of paraylsis are either the result of degenerative processes, hemorrhages or new formations, either at the point of development or somewhere along their course. It is usually not difficult for the practitioner to determine when certain functions are disturbed and a careful examination is in order to determine just how extensive they are and whether more than one of the special nerves might be affected.

One of the very difficult problems in this connection is to determine definitely whether the lesion of the nerve affected is going to be a permanent one or whether the condition producing the paralysis or disturbance is temporary and can be relieved. In observing a large number of these conditions in small animals, we found that it is impossible, in the majority of instances, to establish definitely the degree of injury and, therefore, it becomes necessary first of all to make as positive a diagnosis as possible and then withhold the prognosis until after a few days have elapsed, so as to determine then the extent of the lesion. The majority of these cases which one finds in practice consist of

various degrees of paralysis. Therefore, it is necessary to use stimulants in order to assist nature in overcoming the condition.

Vertigo

There are certain functional diseases which affect the central nervous system and it is necessary to consider them in this connection. Vertigo, which is a condition characterized by dizziness and general disturbance of equilibrium, is found most often in dogs, rabbits, and pigeons. It is generally looked upon as a secondary condition, so far as the central nervous system is concerned. There is a variety of conditions which bring it about, the most important of which are the following: diseases of the brain proper, tumors, hemorrhages of the brain or its membranes, concussion of the brain, difficulties of vision, diseases of the middle or inner ear, sudden changes in the circulation of the blood, such as a dog pulling on the leash, reflex conditions from the digestive tract, ptomaine poisoning, and, in pigeons, an infection which often produces vertigo in large numbers at the same time. In the diagnosis of this condition, we have found that the majority of cases of vertigo come from digestive disturbances and most of them can be corrected by the simple means of obtaining prompt action of the digestive tract.

The most prominent things observed in this condition are a sudden staggering gait, the animal falling down and becoming unconscious, and aften arising in a few minutes seemingly in a normal condition. These attacks of vertigo usually last from two to ten minutes. It is rather difficult in many instances to make a distinction between vertigo and epilepsy. The main difference is the absence of convulsions in vertigo. It seems to me that it is not so difficult to make the diagnosis as it might be to determine the definite cause in all cases. The treatment in these cases should be prompt, putting the patient in a quiet, comfortable place and, as soon as the effect passes away, the animal should be examined promptly to determine the underlying cause and treatment should be advised accordingly. We believe it is always in order in these cases to apply a rather sharp laxative.

Epilepsy

Epilepsy is a disease primarily affecting the central nervous system, characterized by convulsions occurring at irregular intervals and the patient usually being unconscious during the attack. The facts connected with epilepsy, so far as the cause is concerned, have never been established. It seems to be hereditary

in many cases. We make a distinction between true epilepsy, which emanates from some portion of the central nervous system, and reflex epilepsy, which occurs as a symptom of some other disease. There is, however, sufficient difference in regard to the type of symptoms and the length of time the condition lasts to make it rather easy to make the distinction between the two forms. In epilepsy, the attack comes on suddenly, the animal usually falling down and performing uncontrollable movements. Clonic spasms are rather characteristic of this condition. Practically every muscle in the body is in action, including the facial muscles. There is a marked twitching of the facial muscles. Saliva mixed with air flows from the mouth. Often the animal injures its tongue, gums and cheeks. The visible membranes are usually dark and cyanotic; respiration is slow and the heart beats are full and strong.

The convulsion usually lasts only a few moments, the animal gradually becoming quiet and it soon disappears altogether. The animal finally gets up on his feet, staggers about and soon recovers. There seems to be no regularity in regard to the length of time between attacks. There may be one or more per day or per week or per month. We usually advise the owner of the patient affected with this disease that he cannot expect the animal to recover so that attacks will not recur. If treatment is undertaken, the bowels should be kept open by feeding laxative food or occasionally administering a mild laxative. Between the attacks it is well to administer one of the bromids, which seem to produce some results, especially in lengthening the period between attacks. Our experience has shown that the majority of these animals continue to have these attacks at different intervals and death usually ensues during one of them. They may live for several years and only occasionally show the symptom. In reflex epilepsy, when it is observed, the treatment should be directed toward the removal of the primary disease producing it.

Chorea

You no doubt have all observed the condition which is commonly spoken of as chorea, following the nervous form of distemper. You, therefore, I believe, very seldom, if ever, have an opportunity to observe this condition in any animal except the dog. There are three things which seem to produce chorea in this animal. We will mention first, and probably the most important of all, distemper, as chorea is looked upon as a very

common sequel to this disease; myelitis, and the early stages of rachitis in young animals. The diagnosis of this condition is very seldom in doubt, as the prominent symptom of twitching of the muscles is constant, persistent, and continuous. Different groups of muscles may be affected in the different patients. Therefore, it does not matter whether it is the facial muscles, the muscles of the fore limbs, the posterior limbs, or both. Any one of the groups of muscles may be affected. Consciousness is not disturbed in any way. We look upon this condition as chronic and it may persist for months or years. Sometimes, in young animals, recovery takes place without any treatment.

One of the difficult things from a practical standpoint is to state whether or not the individual case at hand will recover. We usually believe that it is unwise to make any prediction in this connection. I think there have been as many different treatments administered in trying to combat this condition as perhaps any of the others that you have to deal with. During recent years we have had the opportunity of observing a number of different drugs which at different times were classified as specific, only to find later on that they had no effect whatsoever upon the disease. Leukocytic extract has been administered lately in this disease, sometimes with apparently good results, but there is considerable question whether this apparent value which has been claimed for it is true or not. Nourishing food and tonics, in the form of iron or arsenic, perhaps are as good as anything which can be administered.

Eclampsia

Eclampsia is a tonoclonic spasm observed in female animals. In the majority of instances it is associated with parturition. As a rule the animal is conscious during the attack of the condition. I do not believe you will find this disease present in any other animal except the dog, at least this has been my observation. The exact cause of the disease has never been found, but it seems to be very closely associated with pregnancy. Perhaps there are certain conditions present, never definitely determined, which produce eclampsia. The disease appears suddenly, usually about ten days to two weeks following parturition and most often it has been observed in small, delicate house-dogs. The animal becomes restless, has an anxious facial expression, later on motor disturbances are seen, the animal falls down and has a spasm of a tonoclonic character. The muscles of the

limbs and body are hard and tense, respiration becomes rapid, pulse slow, the eyes are open, the pupils are normal and the corneal reflex is present. The animal is conscious, the temperature remaining normal or subnormal. The difficulty in diagnosis is to distinguish between this disease and strychnin poisoning. If you consider it from the standpoint that it is a female animal, occurring near the time of parturition and also in the majority of cases that the animal has had no opportunity to obtain strychnin, then the diagnosis is not so difficult. The treatment in these cases should be prompt. Administer morphin or chloroform. Keep the animal quiet and in a warm place. It would be well in all cases to keep the young animals away from the dam. Recovery takes place in the majority of cases in the course of a week or ten days.

There are certain other diseases of this same character affecting the central nervous system more or less directly or indirectly but inasmuch as they do not occur frequently, I do not believe that it is necessary to consider them at this time.

Rabies

There are certain infectious diseases affecting small animals which are of special importance and should be considered in connection with the central nervous system. The first of these is rabies, an acute, infectious disease, fatal in the majority of cases and characterized clinically by disturbance of consciousness, marked irritability and later by symptoms of paralysis. It is not necessary for me to go into any detail in regard to the history of this disease, as it is generally well known. It is well distributed throughout the United States and perhaps during this past year has caused more disturbance and greater losses among all kinds of live stock and the human family than at any time in its history. Statistics show that the disease has been quite widespread during the past six months.

We recognize two forms of rabies clinically, the furious form, affecting the central nervous system primarily and the dumb type, affecting the spinal cord mainly. I do not wish to describe at this time the symptoms of furious rabies as everyone is quite familiar with them. I do wish to call attention to certain things which we find to be of importance from a practical standpoint. First of all, the control of the disease is quite difficult because of its virulence and also it is almost impossible to eliminate the source of infection. It seems to me that the veterinary profession

should take this matter seriously and adopt, if possible, some definite line of procedure, in order to bring about laws stringent enough to control the spread of the infection. I sincerely believe that it would be possible practically to eliminate the disease from this country if certain quarantine regulations could be put into practice, all homeless dogs eliminated, and all others vaccinated at regular intervals.

Inasmuch as this disease is not curable and the diganosis is the important consideration as well as the protection of other animals, I do not think it advisable to go into any further detail in regard to its description. I wish before leaving this subject to caution everyone who has to handle these animals, that any animal showing nervous symptoms, even though it might not be a perfect "book" case, to use every precaution until you are definitely sure that the animal does not have rabies. During the past year we have observed this disease in various forms. We have seen a great many cases of what we would call irregular symptoms and during the early development it was impossible to make a correct diagnosis.

Value of Vaccination Following Bite Questioned

The value of vaccination of animals after they have been bitten by a known rabid dog is questionable. We have had considerable practical experience in the use of vaccine for rabies, using the different kinds, starting years ago with the twenty-one-day treatment and gradually coming down to the present time with a single treatment. We have seen cases of rabies develop after the twenty-one treatments and in practically every other system of treatment used. The percentage of cases developing rabies after vaccination has never been worked out here, but it has led me to believe that it is a question whether we ought to depend on vaccination and lead the owner to have a feeling of security which might not be absolute. I believe, however, that the vaccination of all animals before having been inoculated with the virus will prevent the majority of them taking it later on and, therefore, it should be carried out by the states and municipalities. During the past six months we have vaccinated quite a number of animals, some before being bitten and others afterwards. We have had a few breaks in these cases. Rabies is a disease which has reached the stage where it has become extremely serious and is of vital importance to every veterinarian, dog fancier, and live stock owner. The community should be educated to the seriousness of the condition.

Fright Disease

Fright disease or pseudo-epilepsy has been found in practically all of the states of the Union during the past two years. Formerly this disease was confined almost altogether to the southern states, but I believe that I am safe in saying now that there is no state in the Union that has not had a number of cases of this disease during the past year or so. My experience directly with the disease had extended over a period of about three years. In the autumn of 1922 the first case was presented at our hospital and we were in considerable doubt in regard to the diagnosis. It was not long, however, until a number of other animals in the hospital were affected and we had ample opportunity for making a careful clinical study of the disease as well as autopsy and bacteriological investigation.

We were convinced, after our work in connection with it, that it is a specific infectious disease, having a period of incubation of approximately five to six days and a mortality rate of about five to seven per cent. During this period we were unable to determine anything definite as to the etiological factor, owing to the fact that practically all of the animals of this community were affected or exposed. Various things have been given as the cause of the condition. Various foods on the market have been given credit for distributing the disease throughout the country.

Characteristics of a Specific Infection

Other factors might be enumerated which have been used in connection with the cause of it. All of these things should, of course, be carefully considered, but we believe, owing to its nature, its period of incubation, and the way in which it is spread, that it must belong to the class of highly contagious diseases produced by a specific organism. I am sure most of the practitioners are familiar with the characteristic symptoms, in fact they seem to be so characteristic that when an owner describes the way in which the animal has been acting, the diagnosis can usually be made without even seeing the patient. Sudden excitement, running aimlessly about, barking, the animal showing great fear and excitement, with convulsions in some cases, make a picture which stands out very clearly as compared with any of the other conditions affecting this particular system. We have found in this community that apparently a natural immunity has been established in the majority of animals and we have had only an occasional case of it during the past six months or a year.

Perhaps, in the course of a year or so, it will return in a virulent form again, after the natural immunity of the present generation has run out. There have been a great many drugs recommended in the treatment of this disease. We believe the ordinary sedatives will be as effective as anything that can be administered. Perhaps, in the course of a few years, more will be known about the etiology of it, so that it will be possible to control this disease efficiently and effectively by serum or vaccine treatment or other therapeutic agents.

Lack of time forbids me discussing the nervous form of distemper and certain other diseases affecting the central nervous system.

BUREAU TRANSFERS

Dr. Theodore M. Bayler (Chi. '11), from tuberculosis eradication to meat inspection, Chicago, Ill.

Dr. I. N. Habecker (McK. '08), from tuberculosis eradication to meat inspection, Chicago, Ill.

Dr. E. C. Hughes (Ind. '16), from tuberculosis eradication to meat inspection, Chicago, Ill.

Dr. J. T. Shelton (McK. '18), from tuberculosis eradication to meat inspection, Chicago, Ill.

Dr. H. B. Ballou (O. S. U. '13), from Albany, N. Y., to New York, N. Y., on meat inspection.

Dr. Ellery M. Aldrich (U. P. '13), from Harrisburg, Pa., to Albany, N. Y., on tuberculosis eradication.

Dr. Harry Wende (O. S. U. '15), from Buffalo, N. Y., to Albany, N. Y., on meat inspection.

Dr. Glen C. Harrington (Colo. '23), South St. Joseph, Mo., to South St. Paul, Minn., on meat inspection.

Dr. Clifford L. Hall (U. S. C. V. S. '10), from Portland, Ore., to South St. Paul, Minn., on meat inspection.

Dr. Peter J. Cass (McK. '08), from Madison, Wis., to Milwaukee, Wis., on meat inspection.

Dr. Karl A. Trish (O. S. U. '14), from Milwaukee, Wis., to Madison, Wis., on tuberculosis eradication.

Dr. H. C. Kornetzky (Ind. '21), from Chicago, Ill., to Milwaukee, Wis., on meat inspection.

Dr. M. J. Kemen (Chi. '16), from Chicago, Ill., to Lansing, Mich., on tuberculosis eradication.

Dr. Wm. J. Brown (Chi. '15), from Chicago, Ill., to Mason City, Iowa, on meat inspection.

Dr. John J. Staab (McK. '14), from Chicago, Ill., to Montpelier, Vt., on tuberculosis eradication.

Dr. Francis D. Houston (O. S. U. '15), from Ottumwa, Iowa, to Sioux City, Iowa, on virus-serum control.

Dr. Wm. E. Turner (Chi. '18), from Sioux City, Iowa, to Fort Worth, Texas, on tick eradication.

Dr. John E. Bender (O. S. U. '03), from Fort Worth, Texas, to Nashville, Tenn., on meat inspection.

Dr. Ray O. Porter (K. C. V. C. '09), from Raleigh, N. C., to Lincoln, Nebr., on tuberculosis eradication.

Dr. R. H. Hamner (A. P. I. '19), from Raleigh, N. C., to Atlanta, Ga., on hog cholera control and tuberculosis eradication.

Dr. B. L. Dawson, from Atlanta, Ga., to Raleigh, N. C., on tuberculosis eradication.

SO-CALLED SALMON POISONING IN DOGS*
(Progress Report)

By C. R. DONHAM, B. T. SIMMS *and* F. W. MILLER

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Corvallis, Ore.

Just why salmon should cause symptoms and death when eaten by the dog has long puzzled stockmen and scientists of the Pacific Northwest. In western Oregon, northwestern California, and southwestern Washington, a great many dogs die after having eaten salmon or trout. This trouble apparently does not occur in British Columbia, or Alaska, although both salmon and trout are plentiful in these districts.

HISTORY AND BIBLIOGRAPHY

The history of this disease dates back as far as we have any record of dogs inhabiting this section of the country. There have been numerous theories advanced explaining this problem, but none of them has stood the test of time. There are veterinarians who claim that such a disease does not exist, and that the entire story is a myth, caused by mistaken diagnoses. Many requests for information on this subject come to our laboratory from both veterinarians and dog owners. The above are some of the reasons why we began experimental studies of this disease.

Correspondence with veterinarians brought us very little definite information. Dr. E. F. Pernot,[1] reporting some studies made in Oregon, stated that salmon poisoning in dogs was caused by an amoeba which could be found in the salmon. Calomel was recommended as a curative treatment.

ETIOLOGY

We have found the cause of so-called salmon poisoning in dogs to be a small intestinal fluke, the mature form of which occurs in the intestines of affected animals. Large numbers are present throughout the entire length of the intestinal tract. We have not been able to identify this parasite. Dr. Maurice C. Hall, of the Zoological Division of the Bureau of Animal Industry reports that he cannot yet make a definite statement but is reasonably sure that this is a fluke which hitherto has not been

*Presented at the sixty-second annual meeting of the American Veterinary Medical Association, Portland, Oregon, July 21-24, 1925.

described. It is a distomum, approximately 500 microns in length, and oval in outline. It is hermaphroditic, so each produces eggs. The accompanying microphotograph (figure 1) shows the mature parasite. A cystic form occurs in the muscles, kidney, liver and gills of the fish. The cysts are approximately 140 microns in diameter, and are round. A variation in sizes can be noticed in the same fish. We have definitely demonstrated that this cyst develops into the mature parasite that affects the dog. This was done by feeding fish to dogs and then destroying them at intervals. The length of time required for development of the cysts to maturity is from seven to ten days. Figures 2 and 3 show the cysts in the muscle and kidney of the fish. The suckers can be easily distinguished.

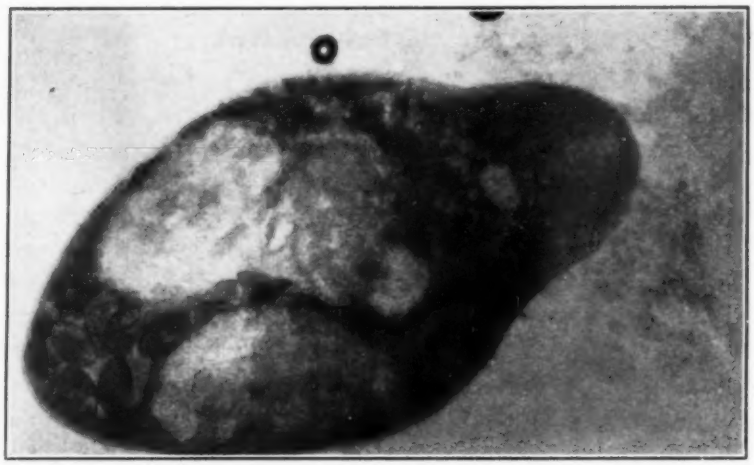

FIG. 1. Microphotograph of mature fluke.

SPECIES OF FISH STUDIED AND GEOGRAPHIC DISTRIBUTION OF THE PARASITE

We have found this cyst in salmon and trout. Salmon are hatched in the fresh water of the mountain streams. After about a year they migrate to the ocean and remain in salt water three or four years and then return to the mountain stream from which they originated. They return to fresh water for spawning purposes, after which act they die. The spawned salmon develop a discoloration of the skin, which has resulted in the popular term of "sore-back" salmon. Cysts are present in salmon before they leave the fresh water and after they return to it. They have not been found in ocean-caught salmon. Susceptible dogs were

not affected by eating ocean-caught salmon. Our first attempts to produce the disease were negative because we were feeding fish that had been caught in salt water. The "sore-back" salmon are severely infested with the cystic form of this parasite. We estimated that there were approximately one million cysts in a pound of severely parasitized muscle of a "sore-back" salmon. The trout may or may not remain in fresh water. We have found these cysts in 121 fish, all of which were caught in fresh water.

Table I gives the number, species and origin of the fish in which the cysts were found. It is a significant fact that these parasites have been found in only the two genera, Salmo and Oncorhynchus.

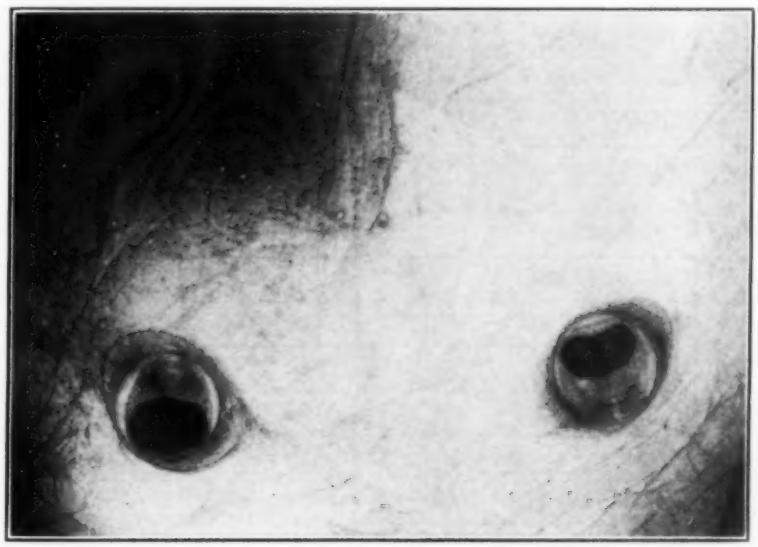

FIG. 2. Cysts in muscle of fish.

One hundred eight fish have been examined in which cysts were not found. Table II gives the data on these. It will be noticed that 64 of them (groups 16 to 22 inclusive, table II) are salmon and trout that came from a fish hatchery. A mature "sore-back" steel-head salmon (group 4, table I) was taken from the same ponds at the same time, and did contain cysts. We cannot explain why these fish that were raised in captivity were not parasitized. The 14 trout (group 7, table II) came from a river east of the Cascade Mountains. The three eastern brook trout (groups 8 and 9, table II) are not of the same genus as the trout reported in table I. The cutthroat trout (group 12, table II)

TABLE I—*Fish in which parasites were found*

GROUP NO.	SPECIES OF FISH	NO. OF FISH	ORIGIN OF FISH
1	Jack salmon	2	Yaquina River tributary, Eddyville, Ore.
2	Steel-head salmon	1	Yaquina River tributary, Eddyville, Ore.
3	Steel-head salmon	6	Nestucca River, Tillamook, Ore.
4	Steel-head salmon	2	Gold Creek hatchery, Tillamook, Ore.
5	Chinook salmon	1	Yaquina River tributary, Eddyville, Ore.
6	Chinook salmon	1	Siuslaw River, Tiernan, Ore.
7	Chinook salmon	1	Nestucca River, Tillamook, Ore.
8	Silverside salmon	21	Yaquina tributary, Eddyville, Ore.
9	Salmon	1	Newport Ice & Fish Co.
10	Salmon minnows	5	Yaquina River tributary, Eddyville, Ore.
11	Cutthroat trout	2	Lake Creek (Siuslaw River) Ore.
12	Cutthroat trout	2	Tillamook, Ore.
13	Cutthroat trout	1	Tillamook, Ore.
14	Cutthroat trout	4	?
15	Cutthroat trout	1	Kilchis River, Tillamook, Ore.
16	Speckled mountain trout	1	Lake Creek (Siuslaw River) Ore.
17	Speckled mountain trout	2	Muddy Creek, Bellefountain, Ore.
18	Speckled mountain trout	6	Tributary of McKenzie River near McKenzie Bridge, Ore.
19	Speckled mountain trout	10	Yaquina River tributary, Eddyville, Ore.
20	Speckled mountain trout	1	Alsea River, Alsea, Ore.
21	Speckled mountain trout	1	Willamette River, Corvallis, Ore.
22	Trout	14	?
23	Trout	10	Alsea River near mouth, Waldport, Ore.
24	Trout	5	Yaquina River tributary, Eddyville, Ore.
25	Trout	20	Triangle Lake, Lane County, Ore.

seems to indicate that this parasite does not infest the fish of the Fraser River in British Columbia. Veterinarians in that section report that so-called salmon poisoning does not occur in their communities. Chinook salmon (group 15, table II) seems to indicate that the fish of the Skagit River in northwestern Washington are not affected with this parasite. Groups 13 and 14, table II, are reported to have been caught in salt water.

TABLE II—*Fish in which parasites were not found*

GROUP NO.	SPECIES OF FISH	NO. OF FISH	ORIGIN OF FISH
1	Whitefish	2	Willamette River near Corvallis, Ore.
2	Smelt	2	Sandy River (Columbia River) Ore.
3	Chub	1	Muddy Creek, Bellefountain, Ore.
4	Sea Bass	1	Netarts Bay, Tillamook, Ore.
5	Silver perch	1	Netarts Bay, Tillamook, Ore.
6	Suckers	5	Umatilla, Ore.
7	Trout	14	Enterprise, Ore.
8	Eastern brook trout	2	Linton Lake (McKenzie Pass)
9	Eastern brook trout	1	East Lake, south of Bend, Ore.
10	Red-side trout	2	Horse Creek (McKenzie River)
11	Red-side trout	1	Tributary of McKenzie River near Bridge
12	Cutthroat trout	1	Cultus Creek, B. C., Canada
13	Salmon—blood only	1	Tide-water, Newport Ice & Fish Co.
14	Salmon	8	Astoria (probably salt water)
15	Chinook salmon	1	Skagit River, Washington
16	Steel-head trout (1 day)	12	Gold Creek hatchery, Trask River, Tillamook, Ore.
17	Steel-head trout (12 days)	12	"
18	Steel-head trout (4 days)	12	"
19	Steel-head trout (1 year)	2	"
20	Cut-throat trout (1 year)	2	"
21	Chinook salmon (4 months	12	"
22	Silverside salmon (6 weeks	12	"

We know that it is a common practice in Alaska to feed dogs salmon as a regular diet. We think that this fluke does not infest fish in the waters north of the Puget Sound country, but we cannot explain this.

Effects of Temperature

Cooking or prolonged freezing of fish destroys the parasites. Cooked salmon did not produce symptoms when fed to dogs. Some parasitized fish was exposed to six days of freezing weather. The temperatures were:

Dec. 22	Maximum 32°F.	Minimum 5°F.
Dec. 23	Maximum 33°F.	Minimum —3°F.
Dec. 24	Maximum 31°F.	Minimum 2°F.
Dec. 25	Maximum 25°F.	Minimum —8°F.
Dec. 26	Maximum 21°F.	Minimum —5°F.
Dec. 27	Maximum 34°F.	Minimum 8°F.

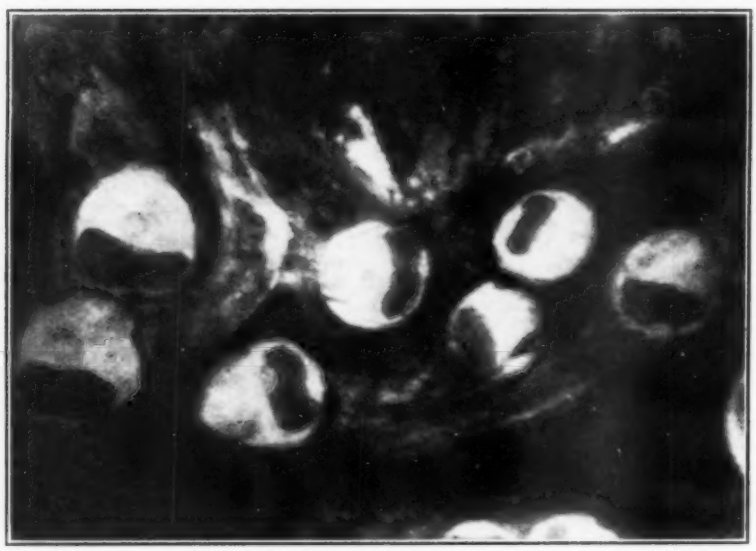

Fig. 3. Cysts in kidney of fish.

This fish had produced the disease before it was exposed to these temperatures, and was harmless to dogs after it had been frozen. The dogs which ate the frozen salmon succumbed after a later feeding of parasitized salmon.

Life Cycle of the Parasite

The complete life cycle of this parasite is not known. We know that the cyst of the fish develops into the mature fluke found in the dog, and that the mature fluke produces eggs which pass out with the fecal material from the dog. Attempts to

hatch the fluke eggs were negative. They were kept at incubation, outside and room temperatures for as long as 90-day periods. We think there probably are other animals which act as the natural hosts of the mature parasite. According to this theory the dog would be classed as an accidental host. It would seem that there are not enough dogs in the coast section of the country to contaminate the water of the mountain streams so grossly that the salmon and trout taken from them would be so severely parasitized. Attempts to find another host of the mature fluke included examinations of the following animals with negative results. We do not know that all of the animals mentioned here had eaten fish recently before examination of their intestinal tracts was made.

Animal	Number	Origin
Squirrel	1	Eddyville, Ore.
Alaska robins	2	Eddyville, Ore.
Crows	3	Eddyville, Ore
Skunk	1	Eddyville, Ore.
Wild cat	1	Eddyville, Ore.
Belted kingfisher	1	Eddyville, Ore.
Chickens (fed parasitized fish)	2	Corvallis, Ore.
Duck (fed parasitized fish)	1	Corvallis, Ore.

Cysts began development in the following animals, but did not reach maturity and produce eggs.

Animal	Number	Origin
Cats (fed parasitized fish)	4	Corvallis, Ore.
Mink	1	Eddyville, Ore.
Raccoon	1	Eddyville, Ore.

We have not been able to examine a coyote. There are very few coyotes inhabiting the section of the country in which we find these parasitized fish. The natives claim that the coyotes are practically all killed by eating the dead "sore-back" salmon that are found along the banks of the small mountain streams during spawning season.

This disease affects foxes as well as dogs. We have had positive diagnoses on two different fox farms. In one instance parasites were found, on autopsy, in our own laboratory. In the other case they were found by Dr. T. B. Carter, of Portland. In both instances these foxes had been fed salmon.

Symptoms

The symptoms do not develop until 7 to 10 days after the dog has eaten the fish. This is the time that is required for the immature fluke to reach maturity. The onset is very sudden. The temperature rises to 105-107° F. This is accompanied by marked

depression, loss of appetite, increased thirst, and in some cases by a discharge of pus from the eyes. There is frequently an edematous swelling of the face, especially around the eyelids. This swelling of the face is probably the most valuable of any of the symptoms in making a diagnosis. These symptoms usually last for 24 to 48 hours, after which the temperature is gradually lowered. At this time a diarrhea develops which is blood-tinged at first, and later is practically all blood. The bloody discharge contains the fluke eggs in great numbers. The animal becomes extremely weak, emaciated, and is frequently unable to stand. In about 6 to 8 days the temperature usually becomes subnormal and death occurs 24 to 48 hours later. An occasional case recovers. Dogs that recover always show these severe symptoms before any improvement is noticed. They are in such an extremely weakened condition that a long period of convalesecnce is necessary.

Diagnosis

The symptoms given above might be found in some forms of dog distemper. It is very likely that this explains some of the controversy as to whether or not so-called salmon poisoning exists as a disease of dogs. We do not think that it is possible to give an absolute differential diagnosis between this trouble and dog distemper from the symptomatology alone.

The history is not a dependable factor in making a diagnosis. The diagnosis can be positively made by finding the parasites on autopsy. The fluke can be seen with the naked eye after the observer is familiar with it. A positive diagnosis can also be made through microscopic detection of the eggs of the fluke in the feces of the affected animal. These eggs are oval in shape, the greatest diameter being about 75 microns. It is usually not very difficult to find the eggs. A small amount of feces is usually sufficient to show them if they are present in any considerable number. If, however, the dog has had a minimum infestation it may be necessary to collect a larger amount of feces and centrifuge it in order to find them. This method of diagnosis has proven satisfactory, and is applicable any time after the symptoms have developed.

Prognosis

The prognosis is always unfavorable. In one experiment we fed one gram of salmon kidney to a dog and produced symptoms and death. We have observed 57 dogs; 34 have died of so-called

salmon poisoning. We have had one dog recover. The balance have been immune, or have died from other causes. We realize that a larger percentage of dogs recover under natural conditions. We can not accurately explain this, but it is probable that a part of the difference comes from most of our dogs receiving a maximum infestation and not receiving good care and nursing.

Immunity

A definite and apparently permanent immunity is present in dogs that recover. Our dog that recovered did not become sick after again eating parasitized fish. We have had nine other immune dogs under observation. Six of these dogs have a history of having been affected and recovered. In addition to these, we have observed numbers of dogs that inhabit the coast country and eat fish regularly. The owners report that such dogs have had the disease and recovered. Attempts to produce and transmit this immunity have been negative. Feeding small numbers of cysts and injecting blood serum from immune dogs were tried. In one experiment we gave blood serum to a dog that had eaten a fish the previous day. In another case, the blood serum was administered the day before symptoms developed. Large doses of blood serum were used as a curative treatment after symptoms developed. In all of these dogs death occurred at the usual time and in the same manner as if the blood serum had not been administered. We can not explain the origin of this immunity. Attempts to demonstrate the presence of a toxin have been negative. The intestinal content of a dog that had succumbed to so-called salmon poisoning was given to a susceptible dog by means of a stomach-tube. This dog remained normal and later died after eating parasitized salmon. The same results were obtained in another experiment, in which a large number of mature flukes were washed, ground up, and administered to a susceptible dog. These experiments with the toxin theory and immunity are too limited to be conclusive.

Negative results were obtained by preliminary bacteriological studies. Only a few of the possibilities of isolating a microorganism have been tried out, however.

Postmortem Findings

The postmortem findings are extremely significant. As has been stated before, the mature flukes and fluke eggs are found in the intestines. There is a marked hemorrhagic inflammation extending from the pylorus of the stomach to the anus. This

inflammation varies in different cases according to the number of parasites present. We have autopsied some immune dogs that had recovered from the disease many months previously, and found a slight hemorrhagic inflammation as described above. This inflammation accompanies the presence of the parasite, but is present where the parasites are not numerous enough to be found. The enteritis is so severe in most cases that blood is found in the lumen of the bowel. There may be a purulent swelling of the ileo-cecal lymph gland.

FIG. 4. Mature fluke in mucous membrane of duodenum.

A study of the microscopic pathology shows that the mature fluke burrows into the mucous membrane of the duodenum (see figure 4). This activity of the parasite seems to be necessary before symptoms develop. Microscopic sections of the intestines have been made in only five autopsies. Three of these have shown this condition. The two dogs in which this was not found both received a minimum infestation. The microscopic sections all show necrosis along the surface of the mucosa. Sections of the kidneys, liver, lymph glands, and lungs have not shown any significant pathology.

Control

No satisfactory method of controlling this disease is known. There have been numerous treatments recommended but none is universally used. We have tried out a considerable number of these treatments, and found them of no merit, at least under experimental conditions. The following drugs have been used as treatment with negative results: calomel, aspidium, carbon tetrachlorid, Epsom salts, castor oil. In addition to the above list we have records of numerous other drugs that have been used by veterinarians, all with indifferent results. It is hard to have faith in any internal medication after we have learned that this parasite imbeds itself in the mucous membrane. Some of the dog owners of the coast country make a practice of feeding fish to small puppies. In this way they are able to find an occasional puppy that will recover. A dog that has not had so-called salmon poisoning is of very little value in this section, while one that is immune to the disease will have a relatively high monetary value, regardless of his breeding. No accurate data as to the number of puppies that recover are available.

Our only recommendation is that the sick dogs be kept warm and comfortable. We have not been able to do this, and all but one of our sick dogs have died. This recommendation, however, will not solve the problem. We have records of a number of dogs which died in spite of good nursing.

Further studies of this problem will be undertaken as soon as time and facilities are available.

Summary

First, the cause of so-called salmon poisoning in dogs and foxes is an intestinal fluke.

Second, the mature parasite occurs in the intestines of affected dogs and foxes.

Third, a cystic form of this parasite occurs in the muscles, kidney, liver and gills of trout and fresh-water salmon.

Fourth, cooking or prolonged freezing of fish destroys the parasite.

Fifth, the mature fluke burrows into the mucosa of the intestine.

Sixth, dogs that recover from so-called salmon poisoning are immune and can eat parasitized fish with impunity.

Seventh, this immunity has not been explained or transmitted.

Eighth, no satisfactory method of treatment or control is known.

Ninth, the complete life cycle of this parasite is not known.

We wish to thank the numerous people who have assisted us in obtaining the material used in these experiments, expecially Dr. J. N. Shaw, of Tillamook, Oregon, and Mr. W. F. Wakefield, of Eddyville, Oregon.

REFERENCE

[1]Pernot, E. F.: Oregon State Board of Health, Bul. 2, Vol. v.

DISCUSSION

MR. T. D. WYATT: I am sorry to say that I am not a veterinarian, but I am studying medicine at the University and, incidentally, I have been working on this disease for the past two or three years. Although Dr. Donham and I have reached quite different results, it is interesting to have both sides of the question. As far as cysts are concerned, I believe, Dr. Donham, you have never found these cysts in salmon in salt water.

DR. DONHAM: No, sir, we have never found them in salt-water salmon.

MR. WYATT: I have been able to trace them in salt as well as fresh; it seems to make no difference whether it was salt-water or fresh-water salmon. I had Dr. Ross get me some "sore-back" salmon from a fresh-water stream, also trout, and the symptoms produced in these particular fish were the same as the ones I found in the salt-water salmon. As far as the pathology is concerned, we do not agree there. I have been unable to find any intestinal condition whatever. There is no hemorrhage. I have any number of slides. I have sections from a number of them. I probably used up four or five tons. I have complete autopsy reports and sections, made by myself and Dr. Benson, who is head of the Pathology Department of the University of Oregon. We were unable to find any intestinal pathology whatever. The pathology is contained in the liver and in the kidneys.

I next made an extract from salmon. This extract was a chemical extract and during the process there could be no possible chance of any life existing, that is, as far as flukes or any bacteria or any form of life. With this extract I was unable to produce salmon poisoning in dogs which went through the same symptoms as dogs with salmon poisoning from the flesh direct. It goes to show that it is not a living organism.

I next tried a process of immunization, and this I have succeeded in doing. I have immunized a number of dogs against salmon poisoning, and to one series of eleven dogs, which was finished last month, I have fed salmon four different times—three different times salmon, and once trout that was caught in a fresh-water stream. The first time it was salmon from the Sound, off Seattle, and the next time it was fresh salmon, that was caught in the Columbia River. The next was "sore-back" salmon, which we got from one of the fish hatcheries, which had spawned there. If there is any chance of flukes whatsoever, it would have been in that case; it smelled bad enough; and the last was trout, and these were fed a month apart, and the symptoms of salmon poisoning did not develop in any one of the dogs.

I next tried purifying the subcutaneous injection of my extract, and I finally succeeded in purifying it so I can give it without causing sores or a condition like that; and in the last series of five dogs I tried to see the exact amount that would immunize the dog. I have succeeded in that, two injections, fourteen days apart, and I have these dogs up at the school now, if any of you would like to see them. You may feed them salmon all you want, and not one of them has had salmon poisoning.

I tried this on a silver fox. Mr. Gains lives out at the edge of Portland here, and I gave him three injections, to be sure it would not kill the fox, because he is worth a little money. We fed him salmon the third time, and he has not shown any symptoms whatever of salmon poisoning. If any of you want to try this out, any group or college, I would certainly be glad to

let you have it. It is not a secret or anything like that. I would be glad to have you try it. I want to be sure my results are right, and as far as I am able to determine, with the help of Dr. Myers and Dr. Benson, at the University of Oregon Medical School, we find no fault with it whatever.

DR. P. G. MACINTOSH: There is a point that seems to be a little confusing to me. Now, up in our country, we have Indians catching salmon, and we all know that an Indian loves his dog, and I have watched them at different times, just out of curiosity. They do not hesitate a minute to feed the dogs the heads and the intestines, everything like that, to the dog, and as far as we could determine, there was not very much loss from feeding this to the dogs. Now, in Alaska, it is one of the main rations of the dogs up there. The point in my mind is that this condition here is isolated to the area, apparently, around the vicinity of the Oregon Coast, distributed through the Northwest. Is this parasite, as spoken of by Dr. Donham, found in this particular salmon in the Columbia River up through our country.

DR. C. A. WHITE: I would like to ask Dr. Donham if he considers this condition transmissible to the human family? We frequently find cases that have had tin-canned salmon, which develop gastro-enteritis and die; usually death takes place about the tenth day. I know of one case, where the family partook of a portion of a can, and fed the balance to cats, and five of them died—five of the cats died, not the family—they were immune. I would like to ask also if it is confined to the salmon and trout. I had a case in Los Angeles where fish had been buried—I believe it was shad—and in the course of a few days, they were dug up by dogs, and three of them, I know, partook of this fish that had been buried for several days, and they all died. In one there was very marked gastro-enteritis. I saw him two days before he died, but I did not detect any edema around the eyes.

DR. R. A. BUTTON: I would like to ask if these eggs were laid after the parasites had entered the mucous membrance, or how soon after the dog had taken them.

MEMBER: I would like to ask if there has been any work done on the intermediate hosts, for instance, eagles, cranes, and pelicans.

DR. SIMMS: In the first place, I want to say that the habit of the salmon is such that he will sometimes be in fresh water and back into brackish water and back in the fresh water again, so it is possible to catch salt-water salmon or salmon just coming out of salt water that will contain these parasites.

I want to say further that when we have had salmon that we knew were salt-water salmon, that carried the lice which they carry in salt water—external parasites, which drop off in fresh water—we have not found these cysts and have not produced the disease. Now, those dogs that die from salmon poisoning, eat fish that contain the cysts. So far as the Columbia River salmon is concerned, that Dr. MacKintosh mentioned, we have no data showing that the parasites occur in this river. We made a very limited examination of salmon taken in this stream, and those limited examinations have been negative. We have no positive evidence of cases of salmon poisoning occurring up in the headwaters of the Columbia, and we know that the salmon do go up into Idaho and into Eastern Washington. We have talked to both veterinarians and stock owners up there who have seen the disease down in the Coast country of Oregon, and they reported they have not seen it in eastern Oregon or in Idaho, where we know salmon do go.

In regard to the question concerning the human, we have no evidence that the human suffers from this trouble. Up to the present, we have not been able to develop the parasite in any animal other than the dog, so we doubt if it will poison any other than the dog family, although we do not know.

In regard to parasitism of the fish, up to the present time we have found this parasite in only two species of the salmon group, including the Chinook salmon, and so on, and the western trout group. Remember that our eastern trout is different from our western trout. The western trout group, including our "cutthroat" and our speckled mountain trout, our "steelhead" trout, are the ones in which we found those parasites. Our examinations of sturgeon and sea bass and two or three other species have been negative. However, they may occur in other species than those studied.

In regard to the length of time after the parasites of the fish are consumed before eggs appeared in the feces, we found it to be approximately seven days. In other words, just about the time the symptoms come on, eggs are being produced and can be found in the feces. The parasite does not necessarily enter the mucosa; the parasite shown on the screen here, for instance, shows the eggs formed in the parasite, and that parasite was obtained in the lumen of the bowels, and had not penetrated the mucous membrance at all.

In regard to intermediate hosts, we feel quite sure that there must be some other host for the mature parasite than the dog; it is unbelievable that we have enough dogs to contaminate our fresh-water streams to such a marked extent, though what that other host is we do not know. We have examined a few birds, and a few of the mammals. Those will be mentioned in the paper that will be published in the JOURNAL. We have examined, for instance, the kingfisher. We have tried to develop the parasite, experimenting with the duck and the fowl, with negative results. I might say further we have not been able to hatch the egg.

We know nothing of the methods of hatching and of the entrance of the parasite into the fish. From our search of medical literature, we know of no complete immunity to parasitic infestation. We do not know that this fluke is the cause of the trouble; it may be the flukes break the mucous membrane and the actual symptoms are produced by bacterial infection.

I am forced to believe that Mr. Wyatt's and Dr. Donham's studies have been of different diseases, because the pathology is entirely different, the hemorrhagic inflammation of the bowel being the one striking thing that we find. Pathologists holding autopsies could not help but find that one striking thing, so I think we have had under consideration two different disturbances.

MEMBER: You stated that a dog can be successfully immunized, and again you say that one gram of the cystic kidney surely will kill any dog. By immunizing that same dog again from that, have you saved one dog?

DR. SIMMS: We have had dogs recover and they have eaten salmon with impunity. In some experiments, we have cut the salmon in two, and fed one-half to an immune dog and the other to a susceptible dog, and the latter died.

We further collected some five or six dogs from stockmen living in the mountains. These stockmen advised us that those dogs were immune to salmon poisoning. They wanted to get rid of them because they were sheep-killers, or something of the sort. In no instance have we been able to produce disease in these dogs. We fed some of those dogs many pounds of salmon. Furthermore, we have recovered the eggs of the fluke from the feces of some of these apparently immune dogs. We autopsied some of these dogs, but we did not find flukes at the time we killed them, nor did we find the parasitic lesions that we always found on our autopsies of the so-called salmon-poisoned dogs.

DR. MACKINTOSH: I would like to ask Dr. Flynn to give us a word on this. He travels all over the continent. Will salmon live in the Missouri River? (Laughter)

DR. J. C. FLYNN: I can give you absolutely nothing on this. This is the first time I have heard this subject discussed. This is the only territory in which I have ever heard of it. I am unfamiliar with it.

DR. BUTTON: I would like to ask Dr. Simms if he has had a chance to use this extract.

DR. SIMMS: We never heard of it until this morning, Doctor.

DR. BUTTON: You have not fed any of those dogs?

DR. SIMMS: No, we did not know what Mr. Wyatt's work was until this morning.

MR. WYATT: If we are working on two different things, I do not see how it could be possible. Why is it when I use simple salmon flesh, we get identically the same symptoms, as far as I am able to determine, by feeding a dog salmon straight and from the extract? How could we be working with two different things?

DR. SIMMS: I think you are working on a different disease, from your description of the pathology. We have in the room here probably thirty or

forty veterinarians who have held autopsies on dogs that died of salmon poisoning. I could point out half a dozen—Dr. Button or Doctor Shaw may be able to tell you the pathology—what they find in the field. When your pathological lesions are so different, you must have a different disease. That is the only conclusion I can reach.

MR. WYATT: The pathology is quite a bit the same, except that the intestinal condition is not.

DR. SIMMS: That is the only thing we find—the intestinal condition. I do not see how they can be the same disease.

MR. WYATT: Produced by salmon, both of them.

DR. O. A. LONGLEY: From the talk presented by the two different groups of men, probably you have a combination of a protein poisoning here with fluke infestation. It would be possible that what was produced in the dogs without intestinal lesions, may be traced to protein poisoning, by using an extract which does not contain any fluke poison and that the fluke could be a factor in producing the intestinal lesion and yet get your protein poisoning just the same. Would it not be possible for you two groups of men to work together?

DR. SIMMS: I, at least, would like to have an opportunity to. This is the first time we have known of Mr. Wyatt's work, except the notice that was in the paper. We have had some correspondence. We have sent Mr. Wyatt's laboratory the results of some of our work. Unfortunately, we did not have a chance to talk with him before today.

DR. LONGLEY: Another thing. You can not produce immunity in protein poisoning, but it is not known that you can produce any form of immunity to fluke infestation. It seems there is a combination of that kind existing in this case.

MR. WYATT: The extract that I prepared is absolutely free from protein.

DR. LONGLEY: Then I have nothing more to say.

MR. WYATT: It had to be free from protein. I would be willing to immunize any dog that Dr. Sims will bring up that has not been immunized whatever, and feed him all the salmon he wants to, and I will buy Dr. Simms a new hat if it will kill him. (Laughter)

TWO MORE LITERARY GEMS

Dr. Edward A. Cahill, vice-president of Pitman-Moore Company, has sent us a letter which certainly deserves a place in our collection of literary gems. Here it is.

1 22 1926

Pittman & Moore Co.

I herd you hanel hoge vaxinaties for hog colare and if you do hanel it let me here from you soon and if you dont hanel and know of eny other ferm dos if you reckmend to me

Anse soon from H—— N——
Edgarton W Va.

Dr. J. H. Bux, state veterinarian of Arkansas, contributes the following "health certificate," recently received at his office.

OlaarkFebruary 3rd 1926

To whom this mau concern:—

This is tocertify that my mules are free from all deceases and free fromall tickets or infection. These are home raised mules and known them from birth.

They have never been exposed to any affection or Malady of any kind.

R. J. Linsey
Notary Public
My commission expires 3/15/26

B. C. Mitchell
Owner

EXPERIMENT WITH MR. THOMAS D. WYATT'S IMMUNIZING MATERIAL AGAINST SO-CALLED SALMON POISONING

By B. T. SIMMS, C. R. DONHAM *and* J. N. SHAW

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On July 31, 1925, some of this material was received from Mr. Wyatt.

Dog 86 was injected subcutaneously with this material, according to Mr. Wyatt's directions, 1.0 cc being given September 8, 1925, and 2.0 cc fourteen days later. This was supposed to produce an immunity in from three to four weeks after the second injection.

Examinations of the feces of this dog for fluke eggs were made for three days previous to the feeding of salmon, with negative results. On November 14 this dog ate *ad lib* a "sore-back" male chum or dog salmon. This fish was obtained from Wilson River, Tillamook County, Oregon. Numerous living cystic flukes were demonstrated microscopically in the kidneys, gills, and muscular tissue of this fish. The dog remained healthy for the next five days.

Typical symptoms of so-called salmon poisoning were observed on November 20, six days after the fish was eaten. Fluke eggs were numerous in the feces at this time. During the following week the disease ran a typical course and the dog died during the night of November 27, 1925.

Autopsy revealed hemorrhagic inflammation of the mucosa of the entire intestinal tube, being most severe in the ileum; myriads of living mature flukes, identical with those previously described as causing so-called salmon poisoning in dogs and foxes, were present throughout the lumen of the intestine; fluke eggs innumerable in intestinal contents; ileo-cecal lymph-gland markedly enlarged and containing pus; cervical lymph-glands slightly enlarged.

No other gross pathological lesions were observed in the lungs, liver, kidneys, or other organs of the body.

Conclusion: Injection of Mr. Wyatt's immunizing material, according to his directions, did not protect this dog against so-called salmon poisoning.

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TOPOGRAPHY OF THE ABDOMINAL VISCERA OF THE OX

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The abdominal viscera of the ox have now been studied in enough cases in this laboratory to indicate, with considerable reliability, their positions and relations. Many of the organs are quite constant in position, while others vary considerably. After noting these variations, one naturally wonders how constant they are and if their occurrence is confined to any particular class of animals.

We believe the first reference to the variable positions of these viscera was given by one of us (Murphey), at the Practitioners' Short Course in Veterinary Medicine at the Iowa State College in 1915.[1] In 1923 Sisson[2] corrected his text-book description of the stomach of the ox with a very good supplemental description. Hopkins[3] described what we consider an extreme case, with no small intestine posterior or ventral to the rumen.

The discussion given here is from a study of our records of observations made on cattle dissected in the laboratory since 1911. Seventy-nine head have been dissected in that time, including fifty-one cows, six heifers under breeding age, four bulls of breeding age, five steers and thirteen bull calves. All were embalmed, by the intravascular injection of formalin, in the normal standing position. These observations have been tabulated and are here summarized. The organs, in systematic order, are briefly discussed, leaving much of the description to the accompanying cuts, as the main object of this article is to add to the literature cuts showing the common variations in position and topography of the abdominal viscera as we have found them. We are using the photographs of two animals which show the two common variations. The photographs were taken at progressive stages of the dissection. The cow was a five-year-old pure-bred Guernsey in good health and eating well when destroyed. The digestive organs were filled with ingesta. The animal was in fair condition.* The bull was a six-year-old pure-

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*The left kidney was found to be merely a vestige; a small renal artery and vein were present but no left ureter could be found. There seemed to be practically no compensatory hypertrophy of the right kidney. It measured 23 x 9 x 7 cm. and weighed 26½ oz.

bred Holstein. He had been ailing for five or six months and was eating but very little when destroyed. However, the only lesions found were slight adhesions of the peritoneum. He was quite thin.

The Rumen

The rumen may fill almost the entire left half of the abdominal cavity, except for space taken by the spleen, reticulum and abomasum. It then extends only a little to the right of the median plane. *However, the ventral sac may be mostly in the right half of the cavity, in which case the intestines fill the ventral part of the left half.*

Dorsal sac: The dorsal sac is the larger. It is always in the upper half of the left side of the cavity, extending from the sixth to the ninth ribs, in front, to a transverse plane through the fifth lumbar vertebra or to the pelvic inlet, behind. It is held against the roof by its attachment from the last rib to the third lumbar vertebra, just to the left of the median plane. The cavity is continued ventrically or ventromedially through a large opening (about 30 cm. x 35 cm.) with the central sac, and communicates anteriorly over a transverse fold with the reticulum. Its chief relations are: dorsally, the sublumbar muscles; laterally, the left abdominal wall, border of the diaphragm and the spleen*; anteriorly, the diaphragm and the reticulum; medially, the omasum, pancreas, left kidney, terminal colon, jejunum, and either the spiral colon or the ventral sac of the rumen; posteriorly, usually the uterus, in females, the small intestine and sometimes the cecum and spiral colon; ventrally, the ventral sac, and sometimes the abomasum in front of the ventral sac, or, when the latter is on the right side, the abomasum, small intestine and occasionally part of the spiral colon.

Ventral sac. The ventral sac of the rumen is shorter than the dorsal sac. It may lie on the floor, largely in the left half of the abdominal cavity, against the lower part of the left abdominal wall, or it may be swung to the right and upward. It then lies on the floor, to the right of the median plane, against the lower right flank, *but in extreme cases it occupies the entire right flank region. Its location on the right is accommodated and always accompanied by a transfer of the movable part of the intestine around the posterior extremity of the rumen, to fill the space otherwise occupied by the ventral sac.*

*In a few cases the upper border of the mass of small intestine may be to the left of the dorsal sac.

When the ventral sac is on the left side, its chief relations are: dorsally, the dorsal sac; to the left, the lower left abdominal wall; anteriorly, the abomasum or the abomasum and reticulum; to the right, the small intestine, colon and usually the omasum; posteriorly, the prepubic abdominal floor and usually some small intestines; ventrally the abomasum, abdominal floor and frequently the small intestines.

When the ventral sac extends to the right flank, its chief relations are: dorsally, the duodenum, first part of the jejunum and parts of the colon; to the left, the dorsal sac, the jejunum and occasionally the spiral colon; anteriorly, the omasum, first part of the duodenum, the abomasum and sometimes the gall-bladder; to the right, the right abdominal wall; posteriorly, usually the uterus, in cows, and the intestine; ventrally, the abdominal floor, usually the abomasum and sometimes the jejunum.

The Reticulum

The reticulum is fairly constant in position. It is mostly in the ventral half of the anterior part of the abdominal cavity, somewhat flattened against the diaphragm. *It is opposite the fifth to eighth intercostal spaces with, usually, from a fourth to a third of its capacity to the right of the median plane. However, it may be entirely to the left of the median plane or it may be about equally divided by the median plane.* The upper part is held in the left half by the esophagus and rumen, but the lower part is free and variable.

Its relations are: dorsally, the diaphragm; to the left, the lower anterior border of the spleen and the border of the diaphragm*; anteriorly, the diaphragm and liver; to the right, it is wedged to a variable extent between the liver, in front, and the omasum, behind; posteriorly, the dorsal sac of the rumen above, the abomasum and omasum and sometimes the ventral sac of the rumen below; ventrally, the abomasum or the abdominal floor.

The Omasum

The omasum is constantly found in the anterior part of the abdominal cavity, mostly to the right of the median plane. It is between transverse planes through the sixth or seventh rib and the tenth or eleventh rib and extending from the abdominal floor† up about one-half to two-thirds of the distance to the

*Occasionally the anterior part of the abomasum is to the left of the ventral part of the reticulum.

†Occasionally the abomasum is directly under the omasum.

vertebrae. While its location is quite constant, its direction and shape vary in different animals.

It is shaped somewhat like a short, thick bean standing on end, with the convex border facing backward and to the right. The opposite border is nearly straight and attached to the reticulum and abomasum, with both of which the omasum communicates. These two openings are about 6 cm. to 10 cm. apart and near the middle of the border.*

Its relations are: dorsally, the liver; on the left, the reticulum, dorsal sac of the rumen, usually the ventral sac of the rumen and the longitudinal part of the abomasum; anteriorly, the liver, the reticulum and the ventral border of the diaphragm; on the right, the liver, gall-bladder, diaphragm and wall; posteriorly, the jejunum, abomasum and sometimes the duodenum, but when the ventral sac of the rumen is on the right side, it displaces all of the jejunum except the first coils; ventrally,† the abdominal floor and the concave border of the abomasum.

The Abomasum

The abomasum is usually either a crescent- or an L-shaped organ, always found on the floor of the abdominal cavity. It curves around the ventral pole of the omasum and may be considered to have a longitudinal or fundus part and a transverse or pyloric part. The fundus is largely to the left of the median plane, extending, usually, from the diaphragm‡ to a transverse plane through the second or third lumbar vertebra and occupying the space from the median plane laterally, to the left, as far as the end of the ninth or tenth rib. Its size may vary from 12 cm. to 25 cm. or more in diameter by 30 cm. to 50 cm. in length. The inlet is at the anterior part of the right border.

The transverse or pyloric part crosses the median plane and lies between transverse planes through the ninth and twelfth ribs respectively. It averages about 12 cm. to 20 cm. in diameter and terminates at the pylorus, which is usually found in the region of the tenth to twelfth costo-chondral joint of the right side.

The relations of the longitudinal part are: dorsally, the reticulum and the ventral sac of the rumen, but when the latter is on the right side, the dorsal sac and sometimes the small intestine; to the left, the diaphragm, abdominal wall or rumen and some-

*The omasum might be ausculted in the region of the costal arch, between the seventh to ninth ribs, sometimes from the sixth to tenth ribs.

†In one case only was the jejunum entirely separated from the omasum.

‡Sometimes the reticulum is between the diaphragm and the abomasum.

times the small intestine; anteriorly, the diaphragm, except when the reticulum separates them; on the right, the omasum and the pyloric part of the abomasum; posteriorly, the ventral sac of the rumen or the small intestine; ventrally, the abdominal floor.

The relations of the transverse part are: dorsally, the small intestine and omasum, but chiefly the ventral sac of the rumen, if it is on the right; anteriorly, the omasum; posteriorly, the small intestine or ventral sac of the rumen; ventrally, the abdominal floor. It is continued medially by the longitudinal part of the abomasum, and laterally by the duodenum.

Sisson states that "the first three parts of the stomach are often regarded as proventriculi or esophageal sacculations, the fourth being the stomach proper."[4] With this in mind, let us note the communications of the compartments. We might consider the esophagus as continuing ventrally from the cardia for about 30 cm. and terminating in the abomasum. This nearly vertical part would then have two slit-like openings, the esophageal groove and the sulcus omasi. The esophageal groove opens on the right wall of the atrium and reticulum. It is a spiral groove, 15 cm. to 20 cm. long, twisted so that the esophageal opening, in the upper end, faces backward into the rumen, toward the last rib on the left side, while the opening into the omasum, at the lower end, faces forward into the reticulum, toward the first ribs on the left.* The groove is located about midway between the tops of the vertebral spines and the xiphoid cartilage, with its upper end opposite the eighth rib or space, usually to the left of the median plane, and its lower end opposite the sixth to seventh spaces, usually to the right of the median plane. The groove is from 1.2 cm. to 2.5 cm. deep and the borders are thick, rounded and in apposition when inactive.

The sulcus omasi commences just below, where the esophageal groove ends, and faces in a nearly opposite direction, backward and to the right into the omasum. The sulcus is not spiral and is more shallow and open than the esophageal groove. It is 6 cm. to 10 cm. long and terminates at the omaso-abomasal orifice.

The Duodenum

The duodenum is constant in its location, being held in place by a peritoneal fold known as the meso-duodenum which continues down over the organ to become the outer layers of the omentum. Thus, the duodenum is usually the only organ

*The position and direction of the lower extremity of the groove varies with the position of the reticulum, which see.

visible when the right flank is opened. It originates from the pylorus, just medial to the costal arch, at about the tenth to twelfth ribs. The first part forms a variable curve,* then continues upward, medial to the tenth to thirteenth ribs, to or below their middle, where it turns backward. The second part continues backward toward the tuber coxae, near which it turns medially to become the third part, which passes forward in the sublumbar region just to the right of the median plane. Under the twelfth or thirteenth thoracic vertebra, the mesentery lengthens and the duodenum is continued by the jejunum.

The first part is usually between the liver and gall-bladder, in front, and the jejunum or rumen, behind; the second part is between the flank, laterally, and the intestines or ventral sac of the rumen, medially; and the third part is under the psoas muscles and right kidney, lateral to the terminal colon and dorsal to the spiral colon or the jejunum. The bile-duct enters near where the duodenum turns backward, which is 35 cm. to 42 cm. from the pylorus. The pancreatic duct usually enters 20 cm. to 30 cm. behind the bile-duct.

The Jejunum and Ileum

The jejunum and ileum are very variable, due to their mobility. They are suspended by a mesentery which encloses the spiral colon in its center, so the whole intestinal mass moves largely as one unit. When the ventral sac of the rumen is on the left side, the intestines are mostly to the right, but when the ventral sac extends to the right side, the intestines change position to accommodate it. The jejunum, being the most movable part of the intestine, then passes around the posterior extremity of the rumen to fill the space, in the lower left flank, made available by the changed position of the ventral sac.

The first few coils of the jejunum are constantly found to the right of the dorsal sac of the rumen, just behind the liver and omasum. The balance is in coils below and behind the first part, but when the ventral sac of the rumen is on the right side, the jejunum passes directly backward, along the longitudinal groove of the rumen, to continue as described above.

When on the right side, the mass of small intestine is surrounded by the part of the omentum which passes from the right longitudinal groove of the rumen to the duodenum. It is therefore separated from the ventral sac of the rumen medially, the

*This curve may be S-shaped, but is not constantly so, as in the horse. It is sometimes a double S and often just a twisted bend.

abdominal floor ventrally, and the right abdominal wall laterally, by the omentum. Its remaining relations are: anteriorly, the omasum and abomasum; dorsally, the colon and usually the duodenum, cecum and pancreas; posteriorly, the prepubic floor, bladder and genital organs.

When on the left side, the relations of the small intestine are: dorsally, the rumen and colon; laterally, the left flank; anteriorly, the rumen and abomasum; to the right, the ventral sac of the rumen (separated by the omentum); posteriorly, the prepubic floor, the cecum and colon or the bladder and genital organs; ventrally, the abdominal floor.

The ileum leaves the border of the mesentery about 38 to 45 cm. before it terminates and passes forward, between the layers of the mesentery at an acute angle with the cecum, to join the large intestine. The mesentery then continues to the cecum, attaching to about the anterior two-thirds of the latter.

The Cecum

The cecum varies much in size and somewhat in shape and position. Being a part of the intestinal mass, it is drawn backward and to the left, when the small intestine is on the left side. Even then it may be curved enough to be entirely to the right of the median plane, or the blind end may be in the left prepubic or flank region. When the small intestine is on the right side, the blind end is usually at the pelvic inlet and the ceco-colic junction is medial to the lower part of the last two ribs. It is usually, then, only slightly curved, but may be the shape of the letter Z or J. The anterior two-thirds is attached by the ileocecal fold; the blind end is free. The relations, when on the right side are: dorsally, the duodenum and colon; medially, the colon and small intestine; ventrally, the small intestine and prepubic floor; laterally, the right flank mostly through the omentum. The cecum is 8 cm. to 13 cm. or more in diameter and from 60 cm. to 75 cm. long.

The Colon

The colon may be divided, for convenience of description, into prespiral, spiral, and postspiral parts. The first is a direct continuation forward of the cecum. It immediately turns and continues backward, medial or dorsal to the cecum, toward the pelvic inlet, then turns under the fourth or fifth lumbar vertebra, to pass forward, under the duodenum, to the right of the rumen.

The spiral starts about medial to the upper part of the last rib, just a little to the right of the median plane. It consists usually of two centripetal and two centrifugal coils, held closely together by areolar tissue and the mesenteric envelope. The spiral forms a body about 38 cm. to 45 cm. long by 15 cm. to 20 cm. wide. It may be drawn behind the rumen or even into the left flank by the other intestines, but when the latter are mostly on the right side, the spiral will also be mostly on the right side. It is then related chiefly to the duodenum and terminal colon dorsally, the rumen medially, and, except for the cecum and prespiral colon laterally, is otherwise nearly surrounded by the small intestine. The postspiral colon leaves the spiral at its anterior end and first takes a turn outward and backward into the angle formed by the prespiral part. Then it passes forward and inward, under the pancreas and portal vein, to beneath the twelfth or thirteenth thoracic vertebra, where it turns backward, as the terminal colon, to join the rectum. The terminal colon* is suspended by a mesentery 6 cm. to 12 cm. wide. It passes backward, a little to the right of the median plane, into which it turns before entering the pelvis.† Its relations are the pancreas, posterior vena cava, right kidney and psoas muscles dorsally; the rumen and the left kidney medially, the third part of the duodenum latero-ventrally, while the other relations are variable.

The Rectum

The rectum is in the upper part of the pelvic cavity and is related ventrally to the bladder, seminal vescicles, prostate, ampullae of the ducti deferentes and pelvic urethra in the bull; to the vagina and vestibule and frequently the body and cervix of the uterus in the cow.

The Liver

The liver is almost entirely to the right of the median plane, with its parietal surface against the diaphragm and the costal part of the abdominal wall and its visceral surface related to the pancreas, duodenum, reticulum, omasum and sometimes the ventral sac of the rumen, jejunum or colon.

The posterior border of the liver usually lies under the thirteenth rib‡ to its middle, then the border curves downward and forward, sometimes to the end of the eleventh rib, then forward

*The attachment and relations of the terminal colon are important for rectal palpation.

†The curve here is very seldom a distinct S shape.

‡In young animals the caudate lobe is large and extends backward under the fourth or fifth lumbar vertebra.

along the costal arch to the xiphoid cartilage. More often the border extends to 8 cm. to 15 cm. from the distal end of the eleventh rib, then it continues forward more or less parallel to the arch, then usually turns down nearly to the xiphoid cartilage in front.* The upper part of the medial border is just to the right of the median plane but below the esophagus the border extends from 2 cm. to 8 cm. to the left, between the diaphragm and reticulum. The gall-bladder, when distended, extends below the border of the liver, medial to the distal ends of the ninth to eleventh ribs. Whether it ends above or below the costal arch depends on the position of the liver and the fullness of the gall-bladder. The bile-duct enters the duodenum about where the second part commences, i. e. 35 cm. to 42 cm. from the pylorus.

The Pancreas

The pancreas is also largely to the right of the median plane, under the upper part of the eleventh rib to the second lumbar vertebra. It is related chiefly to the liver, crura of the diaphragm, right kidney and posterior vena cava dorsally, and to the dorsal sac of the rumen, jejunum and colon ventrally, except when the ventral sac of the rumen comes high enough on the right side to reach it. The portal vein passes through a notch or foramen in the postero-medial border of the pancreas. The anterior border may be in contact with the omasum and the lateral border extends backward along the duodenum enclosed here in the mesoduodenum. Its duct opens into the duodenum about under the last rib, which is from 20 cm. to 30 cm. behind the bile-duct (from 55 cm. to 72 cm. from the pylorus.)

The Spleen

The spleen is constantly found on the left side, with its parietal surface against the diaphragm and its visceral surface against the rumen and reticulum. Its length is usually from 42 cm. to 58 cm. and its average width from 10 cm. to 13 cm. The upper extremity is just below the upper part of the twelfth intercostal space or thirteenth rib, close to the median plane. From there the spleen extends forward, downward and slightly outward, so that the ventral extremity is medial to the ventral part of the sixth or seventh rib. About the upper two-thirds is attached to the dorsal sac of the rumen. The portion related to the reticulum

*The liver shown in the cow is of the latter type; that in the bull is an unusual shape, being greatly reduced ventrally.

varies, but it is usually the lower 7 cm. to 20 cm. of the anterior border.

The Kidneys

The right kidney is constantly found against the sublumbar muscles, under the last rib and the transverse processes of the first three or four lumbar vertebrae. We have found the average size to be about 21 cm. by 9 cm. by 6.2 cm. Its most constant relations are: medially, the right crus of the diaphragm, right adrenal and posterior vena cava; anteriorly, the liver; ventrally and laterally, the pancreas, colon and duodenum.

The left kidney varies in position with the fullness of the rumen* and it is suspended in a fold of peritoneum on the right side of the dorsal sac of the latter. It is usually in or just to the right side of the median plane, under the second to fourth or fifth lumbar vertebrae. Its average size is 20 cm. by 7.5 cm. by 10 cm.

It is constantly related to the dorsal sac of the rumen on the left, to the terminal colon on the right and usually to the spiral colon ventrally. The anterior pole may be in contact with the left adrenal, but usually there is a space between the two.

Discussion

The female genital organs have not been discussed, even though they extend into the abdominal cavity. We feel that they merit a more careful and detailed description, which will appear in a later article.

In all newborn calves the rumen is small and flattened against the left abdominal wall. Frequently it does not extend below or behind the costal arch, and the small intestine then fills the left flank. In older calves the rumen extends along the left flank to the left of the small intestines. The left kidney lies just to the left of the median plane against the psoas muscles. We presume that the rumen develops against the left abdominal wall, displacing the left kidney and intestines as it enlarges, since we have found it in this position in most males and in the females which had not been pregnant. In cows which have been pregnant the ventral sac of the rumen is frequently over against the right flank and the small intestine is then on the left side. We therefore assume that pregnancy is a factor in placing the organs in the latter positions. However, we have some evidence that the rumen may develop to the right, dorsal to the intestinal mass,

*In one cow the left kidney was fixed dorsal to the rumen.

instead of downward and to the left of the latter. More observations are necessary before a definite statement can be made.

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KEY TO LEGEND

(Figures 1-13)

a, abomasum; ao, aorta; c, colon; ce, cecum; co, origin of colon; ct, terminal colon; d, duodenum; di, diaphragm; e, esophagus; eg, esophageal groove; f, fat; g, gall-bladder; h, heart; j, origin of jejunum; im, intercostal muscles; k, right kidney; l, liver; m, mesoduodenum; md, mediastinum; o, omasum; om, omentum; p, pancreas; pvc, posterior vena cava; py, pylorus; r, reticulum; rd, rumen, dorsal sac; rv, rumen, ventral sac; rr, rumeno-reticular fold; ap, anterior pillar; pp, posterior pillar; cp, coronary pillar; bs, dorsal, posterior blind sac; s, small intestine; sp, spleen; t, trachea; v, subcutaneous abdominal vein; vp, portal vein; x, right lung; y, left lung; z, pericardium; 1-13, respective ribs or costal cartilages; 14, scapula or scapular cartilage.

(Numbers in parentheses following numbers of figures refer to photographs and other illustrations in gross anatomy previously published from this laboratory.—Authors.)

FIG. 1 (134) Right side of Holstein bull with thoracic and abdominal walls removed.

FIG. 2. (135) Right side of Guernsey cow. (Dissection similar to fig. 1)

FIG. 3. (136) Same as fig. 1 but with diaphragm and omentum removed.

FIG. 4. (137) Same as fig. 2 but with diaphragm and omentum removed.

FIG. 5. (138) Same view as fig. 3 with lung and part of liver removed. Arrow points to the bile-duct entering duodenum.

FIG. 6. (139) Cow, same view as fig. 4 but with lung and liver removed. Arrow points to the bile-duct.

FIG. 7. (140) Left view of bull. Gas has escaped from the dorsal sac of the rumen and permitted it to draw away from the abdominal roof. Some adhesions are seen posteriorly

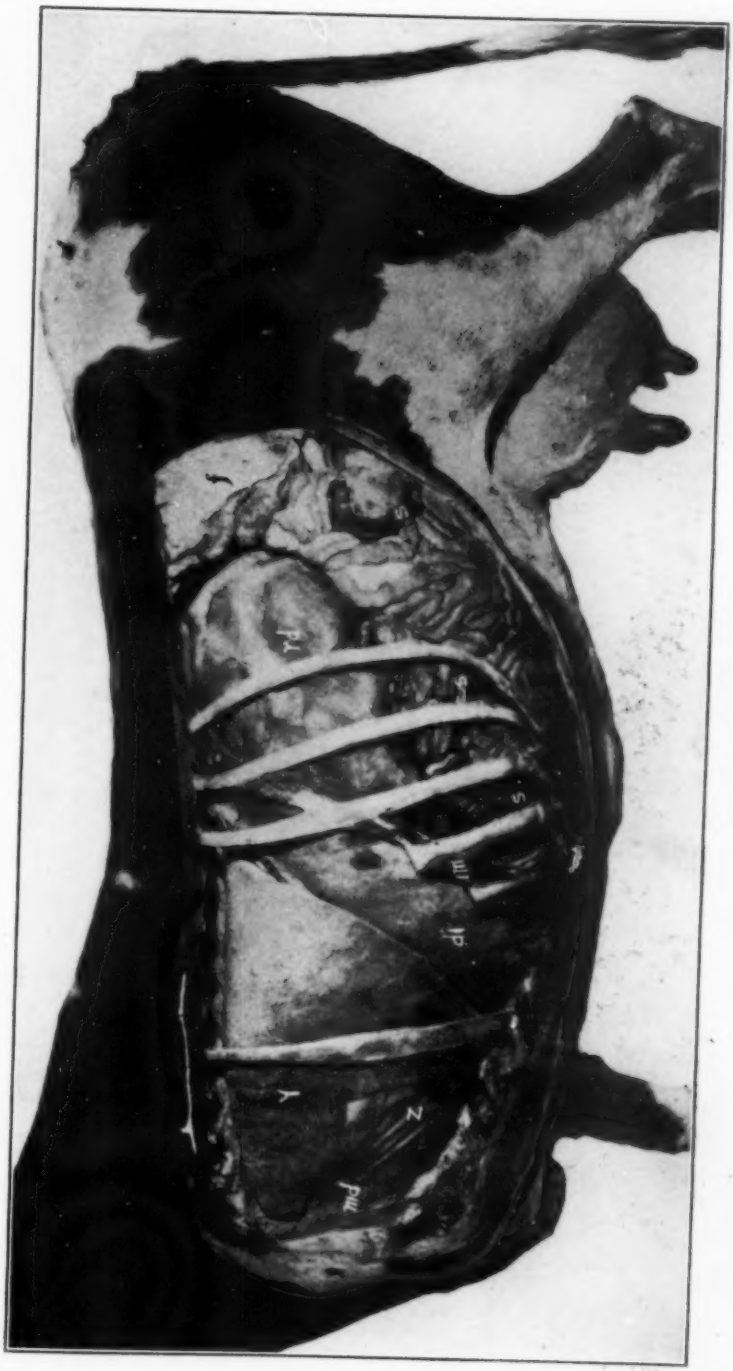

FIG. 8. (141) Left view of cow.

FIG. 9. (142) Same as fig. 7 but with left lung, part of the diaphragm and part of the spleen removed.

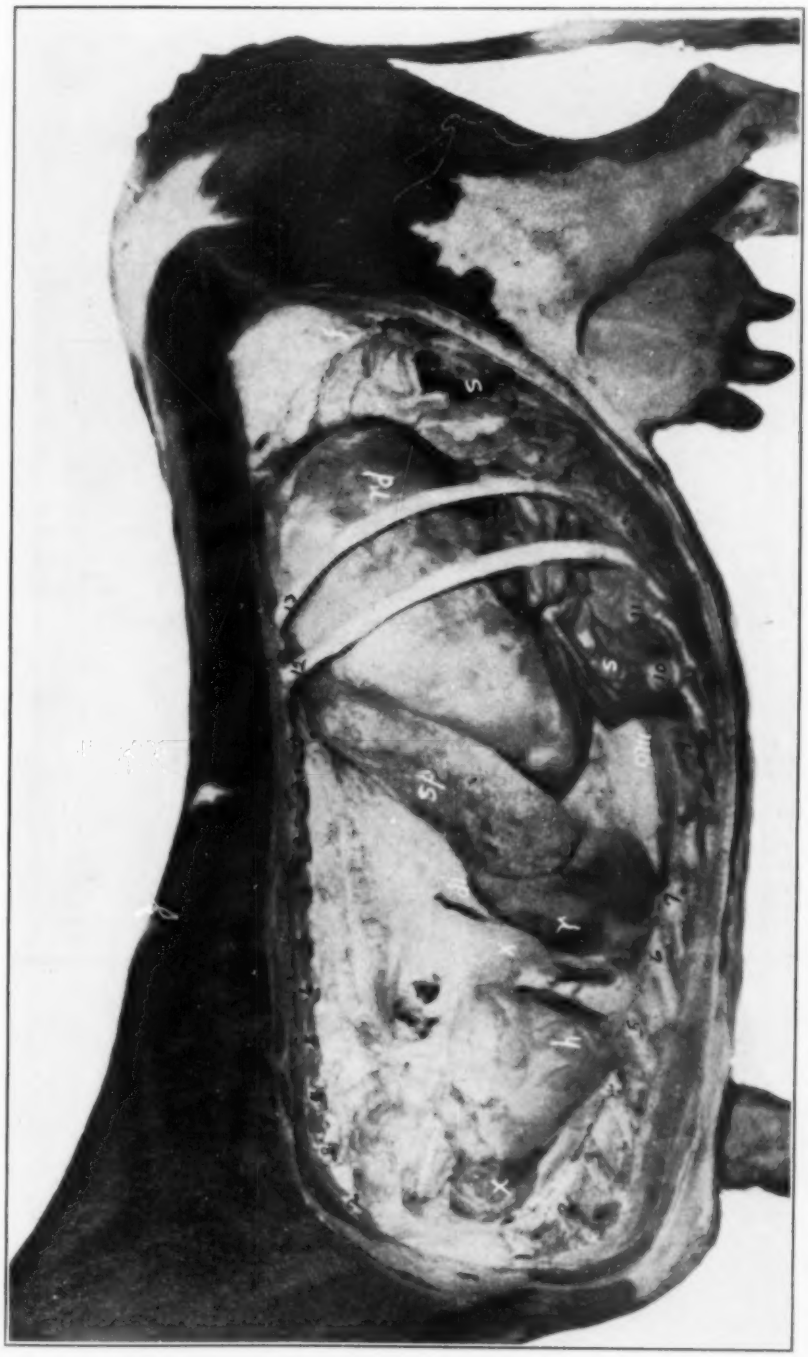

FIG. 10. (143) Same as fig. 8 but with the left lung and part of diaphragm removed. The omentum contains a mass of fat which covers the abomasum.

FIG. 11. (144) Same as fig. 9 but with rumen and reticulum opened from the left side. A rubber tube extends through the cardia.

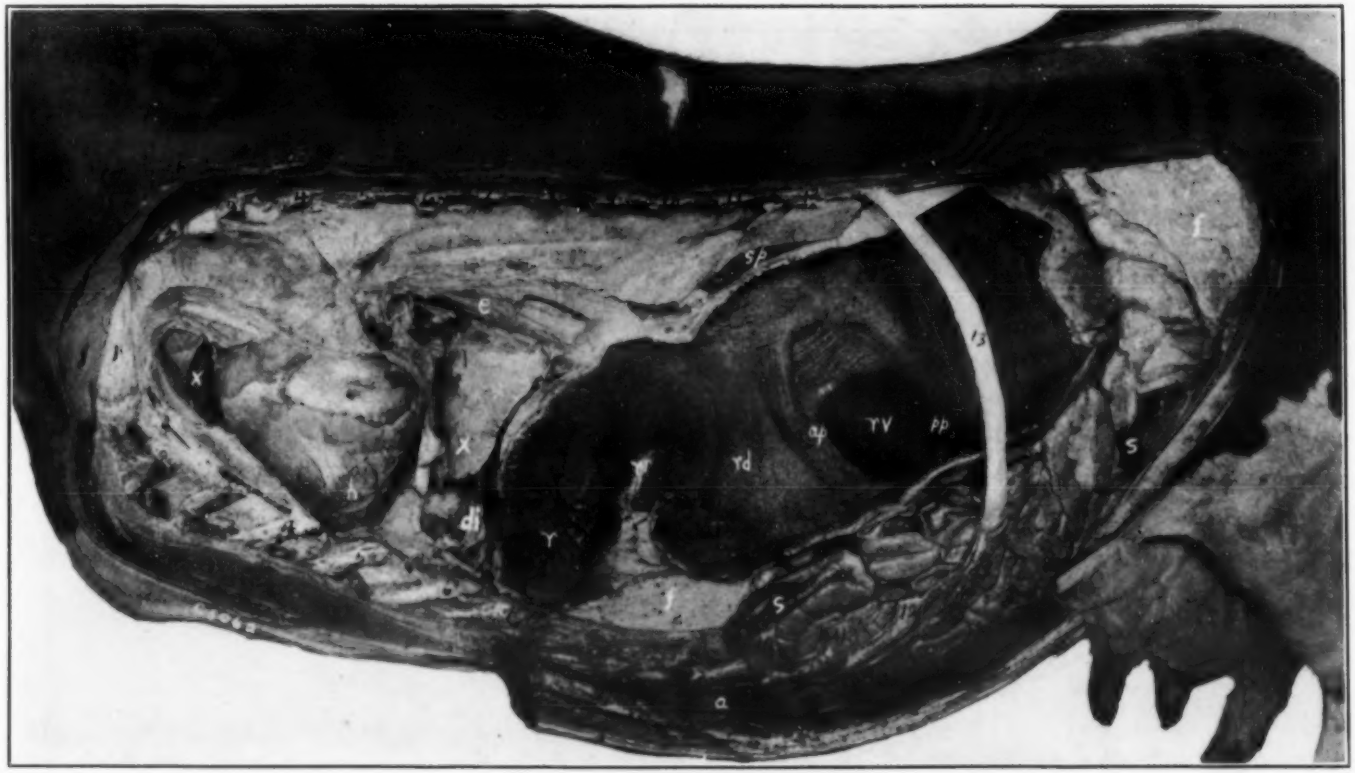

FIG. 12. (145) Same view as fig. 10 but with spleen, lateral wall of reticulum and lateral wall of dorsal sac of rumen removed. It was taken after fig. 13 and the organs have unavoidably dropped several inches, the reticulum being drawn backward. Note the contrast with fig. 11. A section of the wall of the esophagus has been removed and the esophageal groove is kept open by two blocks of wood.

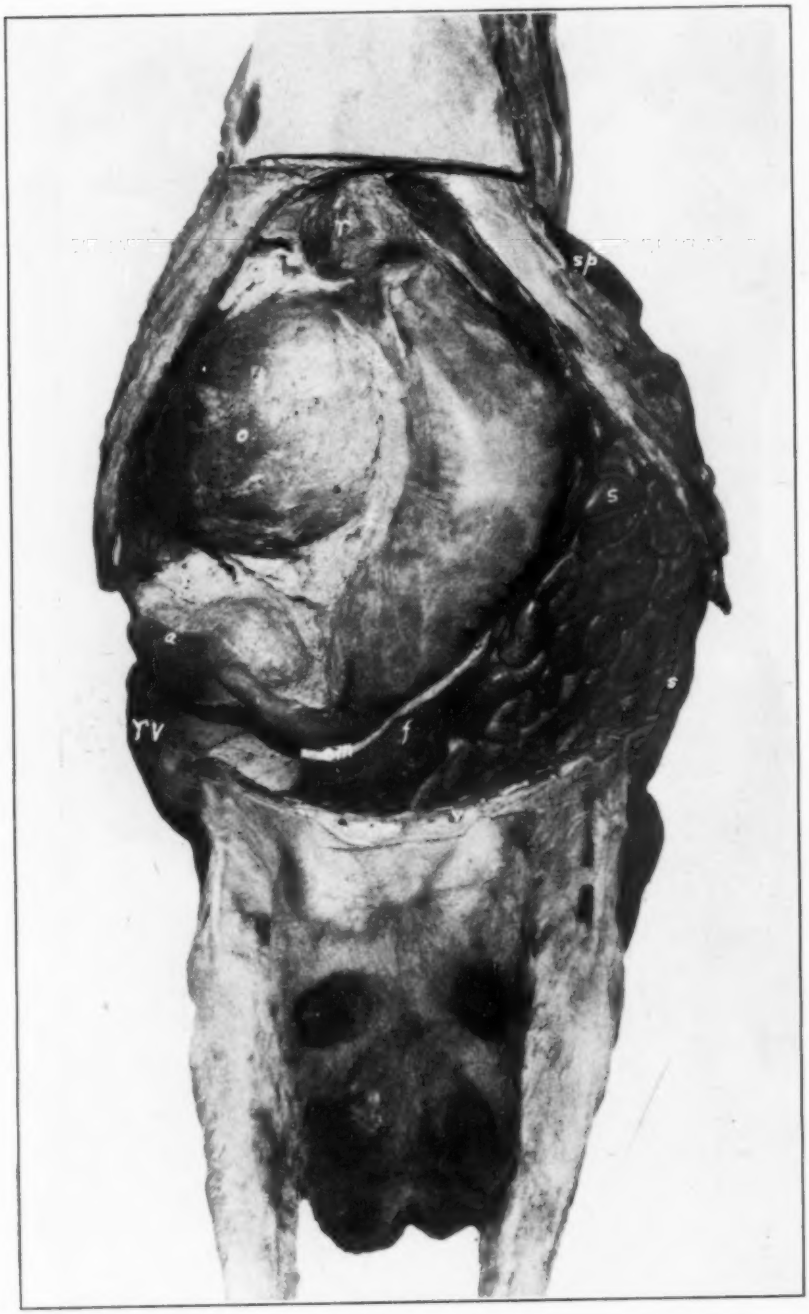

FIG. 13. (146) Ventral view of cow, omentum removed. There is no appreciable displacement of the organs.

MISCELLANEOUS TESTS OF TETRACHLORETHYLENE, A NEW ANTHELMINTIC*

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As a result of their work on the use of various chlorin compounds, for the removal of hookworms, Hall and Shillinger[1] recently recommended the cautious use of tetrachlorethylene (C_2Cl_4) for the removal of these parasites from man.

This recommendation was based on the results obtained following the use of this compound in dogs, a detailed account of which, and the recommendation for its use for removal of hookworms from these animals were given in a later publication.[2]

Afer completing a number of tests on puppies, the author[3] also found that this new anthelmintic was as efficient as and possessed a greater factor of safety than did carbon tetrachlorid (CCl_4), a closely related compound which has enjoyed a widespread use for the removal of hookworms, from both man and animals, since Hall[4] first recommended it for that purpose in 1921.

Keeping in mind its efficiency against hookworms and its greater factor of safety for puppies especially, tests were made with tetrachlorethylene to determine its effect on the various internal parasites, and on the hosts themselves, when given to animals of various species.

With the exception of the dogs described later in this publication, none of the animals was given a purgative simultaneously with or following the administration of tetrachlorethylene.

Throughout the entire series of experiments the anthelmintic was given in soluble elastic globules, except to horses, to which it was given in hard gelatin capsules. Globules of 0.2-cc, 1-cc and 5-cc capacity were used.

TESTS ON DOGS

In the tests of tetrachlorethylene previously reported by the author,* this new anthelmintic was given to all dogs in hard gelatin capsules.

To determine its effectiveness when given in soluble elastic globules, six adult dogs were used (table I), the anthelmintic

*Read before the Southeastern Michigan Veterinary Medical Association, Detroit, Mich., December 16, 1925.

being given, for practical purposes, at the rate of 1 cc for each 10 pounds of body weight or fraction thereof, which was equivalent to approximately 0.2 cc per kg.

In these tests the anthelmintic had no effect on tapeworms (dog 145) or on whipworms (dog 71). It was, however, 100 per cent efficient against roundworms and hookworms where these parasites were present.

Dogs 71 and 18 were used on account of suffering from mange at the time of treatment. Death of dog 71 occurred two days after the anthelmintic was given. Postmortem examination showed no apparent macroscopic changes in the internal organs. Dog 18 showed no toxic symptoms during the four days it was under observation, and showed no apparent changes in the

TABLE I—*Tetrachlorethylene in soluble elastic globules to dogs at the rate of 1 cc per 10 pounds of body weight or fraction thereof*

Dog	Wt. (lbs.)	C_2Cl_4 (cc)	Worms Passed		Worms Postmortem			
			Round	Hook	Round	Hook	Tape	Whip
145	25	3.0	0	0	0	0	5	0
71	22	2.0	31	4	0	0	0	77
25	44	5.0	5	1	0	0	0	0
31	18	2.0	0	1	0	0	0	0
35	25	3.0	0	2	0	0	0	0
18	42	4.0	0	0	0	0	0	0

internal organs on autopsy, as was the case in the remaining dogs of this group.

While one of these dogs, suffering from mange, showed no toxic effects from the drug, it would seem nevertheless, that, since the other one died within two days, the drug would, as Hall and Shillinger[2] suggested, be contraindicated in animals suffering from such a condition.

In their original work Hall and Shillinger found that the simultaneous administration of a purgative with tetrachlorethylene had a tendency to diminish its anthelmintic efficiency. They suggested that the administration of a purgative an hour or more after the treatment might give better anthelmintic results with about the same safety.

To determine what effect this later administration of a purgative might have, four dogs (table II) were given tetrachlor-

ethylene, followed in from one to two hours by dry Epsom salts, enclosed in No. 11 hard gelatin capsules which held approximately 5.5 gms. each. The salts were given to the first three dogs two hours after the tetrachlorethylene, the dose of which was at the rate of 1 cc for each 10 pounds of body weight or fraction thereof. Dog 4 received salts one hour after the anthelmintic.

From the results of this test it would seem that tetrachlorethylene, in soluble elastic globules, followed in less than two hours (dog 4) by dry Epsom salts, was possibly reduced somewhat in efficiency. These results were probably produced by the purgative removing the anthelmintic or a portion of it, before it had its full action on the parasite, as the odor of the tetrachlor-

TABLE II—*Effect of tetrachlorethylene in soluble elastic globules followed by dry Epsom salts*

Dog	Wt. (lbs.)	C_2Cl_4 (cc)	Time Between Doses (hrs.)	Worms Passed		Worms Postmortem	
				Round	Hook	Round	Tape
1	22	3.0	2	15	1	0	0
2	21	3.0	2	4	0	0	0
3	28	3.0	2	15	5	0	0
4	26	3.0	1	3	0	2	12

ethylene was noted in the first passage of feces from this dog, a condition not encountered in other animals following the administration of a therapeutic dose.

To obtain information as to the effect of a therapeutic dose of tetrachlorethylene on a pregnant bitch, the anthelmintic was given to one showing evidence of approaching parturition. As in the case of the other dogs, this bitch, which weighed 58 pounds, was fasted overnight before 6 cc of tetrachlorethylene was given. Within 48 hours, eight living normal puppies were born, labor having begun about 24 hours after administration of the anthelmintic. At no time during the ten days this animal was under observation were any untoward symptoms noted. The puppies were all normal at this time. No autopsy was made. It was not thought that the administration of tetrachlorethylene influenced the time of parturition in any manner.

Tests on Chickens

To determine the effect of tetrachlorethylene on chickens, twelve Leghorn hens were used (table III). Their weights varied from 0.91 kg. to 1.6 kg. The anthelmintic was given in doses varying from 1 cc to 5 cc. No difficulty was experienced in giving the larger globules to these birds.

Birds 1 and 2 were autopsied on the third day after receiving tetrachlorethylene; birds 3 and 4 on the fourth day, while birds 5, 6, 8, 9, 10 and 11 were autopsied on the fourteenth day. All

Table III—*Tetrachlorethylene to chickens in doses of 1 cc to 5 cc*

No.	Weight (kg.)	C_2Cl_4 (cc)	Worms Passed	Worms Postmortem
1	1.32	1.0	0	0
2	1.08	2.0	0	0
3	1.00	1.0	0	0
4	1.32	2.0	0	0
5	1.30	3.0	0	0
6	1.00	4.0	0	0
7	1.20	5.0	0	0
8	1.20	1.0	1†	1†
9	1.60	2.0	1*	0
10	1.00	3.0	1†	0
11	0.91	4.0	0	0
12	1.12	5.0	0	0

†Roundworm. *Tapeworm.

of these birds were in good condition at the time the postmortems were held and showed no lesions which were thought to have been due to the effect of the anthelmintic. A slight yellowish cast of the livers was noted, which may have been due to fatty infiltration from natural causes. No toxic symptoms were noted during the time these birds were under observation.

Only three of these birds (8, 9 and 10) were infested with internal parasites. One cc of tetrachlorethylene removed one of the two intestinal roundworms present in bird 8, the remaining one being dead when postmortem examination was made. Two

cc caused the removal of the one tapeworm from bird 9, while three cc removed the one intestinal roundworm from bird 10.

During the four days after 5 cc of tetrachlorethylene had been given to birds 7 and 12, no toxic symptoms were noted. At the end of this time, bird 7 was given 15 cc and bird 12 was given 10 cc. Forty-eight hours later, this latter bird was noticeably dull, the appetite being somewhat impaired. On autopsy, seven days later, the intestines were found to be empty except for a small amount of bile. The liver was lighter in color than normal and rather friable. The gall-bladder was filled with very thick, dark-green bile. The gizzard was filled with food mixed with bile.

Four days after bird 7 was given 15 cc of tetrachlorethylene it refused all food and was extremely dull, but on the seventh day, when it was destroyed, it had shown slight signs of improvement. During the last two days this bird was allowed to live, a rather profuse greenish-colored diarrhea was present. On autopsy it was seen to be emaciated and, on opening the peritoneal cavity, a faint odor of tetrachlorethylene was noted. The liver was somewhat lighter in color than normal. The gall-bladder was distended with thick, dark-green bile. The intestinal tract was empty and showed no sign of inflammation.

From these results it is apparent that tetrachlorethylene in doses of from 1 cc to 5 cc, or at the rate of approximately 1 cc to 5 cc per kg., is not toxic to adult chickens.

A repetition of 10 cc or 15 cc to birds which had previously received 5 cc caused toxic symptoms as shown by dullness, impaired appetite and diarrhea. These larger doses caused some liver changes, while those following the smaller doses were very slight, if any, on macroscopic examination. The results of microscopic examinations made of sections of the livers, kidneys, and spleens of these birds, as well as examinations of these organs from all the animals used in these tests and upon which post-mortems were held, will be reported in a later publication.

The minimum efficient dose for chickens is probably not more than 3 cc nor less than 1 cc. Further work on heavily infested birds is being done to determine this dosage.

The removal of the one tapeworm from bird 9, following the administration of 2 cc of tetrachlorethylene, is not conclusive evidence that this drug is effective against the tapeworms of chickens. Considering its action on the tapeworms of animals,

one would conclude that the drug has no anthelmintic action against tapeworms of chickens.

Tests on Cats

A half-grown cat weighing 3.5 pounds was given 0.4 cc of tetrachlorethylene (table IV), the dose rate being approximately 0.2 cc per kg. An aged cat weighing 6 pounds received 1.4 cc of the anthelmintic which was at the approximate rate of 0.5 cc per kg.

During the four days these cats were under observation no toxic symptoms were noted. The anthelmintic in the doses given was 100 per cent efficient against roundworms and hookworms in the first case and against roundworms in the second case.

No abnormalities were noted on postmortem of either animal, except possibly slight evidence that there had been some degeneration of the liver of the cat which received the larger dose.

Table IV—*Tetrachlorethylene to cats in doses from 0.4 cc to 1.4 cc*

Cat	Weight (lbs.)	C_2Cl_4 (cc.)	Worms Passed		Worms Postmortem
			Round	Hook	
1	3.5	0.4	13	8	0
2	6.0	1.4	9	0	0

It would seem that the dose rate as recommended for dogs, i. e., 0.2 cc per kg., is sufficient to remove roundworms and hookworms from cats and can be given to these animals with safety.

Tests on Foxes

Through the courtesy of Mr. F. W. Crafts, of the Detroit Silver Fox Farms, Pontiac, Michigan, and with the cooperation of Dr. S. R. Johnson, pathologist, Michigan Department of Agriculture, and Dr. F. E. Morgan, field veterinarian, tests with this anthelmintic were made on seven foxes (table V). The doses ranged from one to ten cc. These animals were held under observation for six days with the exception of the last two, which were destroyed on the fourth day. These two animals were treated and autopsied by Dr. Johnson who reported that no abnormal postmortem lesions were found, other than a slight degeneration of the liver in fox 7, nor had there been any evidence of toxicity shown while they were under observation. Fox 7

in reality received more than 10 cc, since, when the second 5-cc globule was being given, it was crushed between the teeth and a portion of the contents escaped into the mouth. The globule, with the remainder of the tetrachlorethylene, was removed from the mouth and another given in its place. No evidence of irritation to the buccal mucous membrane or of asphyxiation was noted.

None of the other foxes showed any abnormal symptoms at any time, nor were any abnormal lesions noted on autopsy. Fox 1 passed no worms nor were any found postmortem, although on preliminary fecal examination hookworm ova were found. Undoubtedly, the animal was infested with hookworms and the treatment was 100 per cent efficient. Hall[4] and Hanson and Van

TABLE V—*Tetrachlorethylene to foxes in doses of 1 cc to 10 cc*

FOX	AGE	WEIGHT (LBS.)	C_2Cl_4 (CC)	WORMS PASSED		WORMS POSTMORTEM
				ROUND	HOOK	
1	3 yrs.	8.50	1.0	0	0	0
2	3 yrs.	7.50	1.0	0	2	0
3	6 mos.	6.00	1.0	1	1	0
4	3 yrs.	8.50	2.0	1	0	0
5	3 yrs.	8.25	3.0	0	1	0
6	9 mos.	6.50	5.0	3	28	0
7	3 yrs.	7.50	10.0	0	38	0

Volkenberg[7] report similar experiences. The dose rate recommended for dogs seems approximately correct for foxes.

Comparing the results obtained in adult foxes with those obtained in dogs and knowing the great margin of safety of tetrachlorethylene for dog pups, the judicious use of this anthelmintic in fox pups should not be followed by any untoward symptoms. Further trials are necessary, however, to determine its limitations when used in these young animals.

Of exceptional interest regarding the effects of accidental breakage of the globules, and the subsequent escape of tetrachlorethylene into the upper respiratory tract, was an accident which occurred while giving the globules to fox 4. After moistening the globules with water, the first one was given without any difficulty. When the second was discharged from the balling-

gun into the pharynx it became lodged there, pressing on the epiglottis, causing a complete occlusion of air. By pressure on the throat, the capsule was broken and dislodged, allowing the contents to escape. As soon as the broken globule was removed and a slight amount of artificial respiration resorted to, the fox revived completely.

It was apparent that these alarming symptoms were due to mechanical obstruction of the larynx and not to the tetrachlorethylene, since no further symptoms were noted. No doubt, accidents of this sort can be prevented if the globules would be coated with neutral oil immediately before administration.

The diet of these animals was not changed either before or after giving the anthelmintic. It consisted of beef hearts, ground bone (green), milk, carrots, fox biscuits and cod-liver oil.

TABLE VI—*Tetrachlorethylene to calves, 1½ years of age, in doses of 15 cc to 35 cc*

CALF	WEIGHT (LBS.)	C_2Cl_4 (CC.)	WORMS PASSED	WORMS POSTMORTEM
3943	376	15.	0	0
4057	376	20.	0	0
3938	384	25.	0	0
4055	420	30.	0	0
4035	480	35.	4*	8*
4051	430	Control	0	10*

*Whipworms.

As a result of these tests, one would be led to believe that the administration of therapeutic doses of tetrachlorethylene to foxes would not be followed by symptoms of delayed intoxication such as are described by Hanson and Van Volkenberg[6] following the use of carbon tetrachlorid. However, it would be advisable to bear in mind the conditions in which tetrachlorethylene would be contraindicated and to leave untreated those animals which were suffering from such conditions.

TESTS ON CATTLE

In these tests there were used five native Michigan calves, each aged about one and one-half years and varying in weight from 376 to 480 pounds (table VI). The most unthrifty of those available were selected for the tests, which were begun after withholding food for 24 hours.

The doses of tetrchlorethylene varied from 15 cc to 35 cc. These amounts of the drug at no time produced any untoward symptoms during the seven days the calves were under observation. During this time, no worms were found in the feces from any of these animals except in those from calf 4035, where four whipworms were found. On postmortem, eight whipworms were present in the cecum of this animal, while no worms of any sort were found in the others.

All organs were apparently normal, appearing no different than those from calf 4051 which was received from the same source with the treated ones and slaughtered at the same time, but which had received no tetrachlorethylene. Ten whipworms were found in the cecum of this animal.

While the results of these tests did not show the effectiveness of tetrachlorethylene for hookworms and roundworms of these

TABLE VII—*Tetrachlorethylene to horses in doses of 0.066 cc to 0.15 cc per kg.*

HORSE	WEIGHT (LBS.)	C_2Cl_4 (CC PER KG.)	TOTAL CC	WORMS PASSED		WORMS POSTMORTEM		
				BOTS	PIN	BOTS	PIN	ROUND
910	980	0.066	29.3	8	0	10	0	0
912	1040	0.10	47.0	0	0	0	0	0
911	1120	0.15	76.0	14	1	18	18	1

animals, they did show that this anthelmintic can be given to calves, such as were described, in doses of 15 cc to 35 cc, without danger.

Possessing a marked effectiveness against hookworms and roundworms of other animals, tetrachlorethylene should prove effective against these parasites in cattle, the doses being probably 15 cc for yearlings and 32 cc for animals weighing 700 to 800 pounds, as for carbon tetrachlorid.

TESTS ON HORSES

Three aged horses, weighing 980 pounds (445 kg.), 1040 pounds (472 kg.) and 1120 pounds (509 kg.), respectively, were used for these tests (table VII). Tetracholrethylene was given at the rate of 0.066 cc, 0.1 cc and 0.15 cc per kg. to these animals in the order named, about six hours after a light feed.

About one and one-half hours after the anthelmintic was given, horse 910 showed symptoms of drowsiness, standing with its

chin resting firmly in the feed trough. When moved, there was a marked incoordination of the muscles, especially those of the hind limbs. The symptoms shown were not sufficiently marked to cause the animal to fall or lie down but passed off in about three hours. Only a small amount of feed was eaten that night, but next morning the horse had regained its usual appetite and continued to be apparently normal throughout the time it was under observation.

Horse 911 showed only very slight symptoms of drowsiness, which passed off in about one hour. Horse 912 showed these symptoms to only such a slight degree that they were barely noticeable. No other symptoms were shown. On the fourth day these horses were destroyed and autopsied. No lesions thought to have been due to the effects of tetrachlorethylene were found.

None of these horses passed any roundworms, only one being found postmortem (911). The anthelmintic in the doses given was about 50 per cent effective against bots. It might have appeared more so if the animals had been kept under observation longer, for, as Hall, Smead and Wolf[5] found, following the administration of carbon bisulphid, the bots may have remained dead in the intestinal tract for a longer period than four days. Of the ten bots found postmortem in horse 910, only four were attached to the wall of the stomach, the remaining six being dead and free in the small intestines. Only one of 19 pinworms was passed by horse 911.

From these tests it is evident that tetrachlorethylene can be given to horses in doses up to 0.15 cc per kg. without any exceedingly harmful effects, these being only transient. Since these animals were all extremely aged, the symptoms shown shortly after receiving the anthelmintic may possibly have been due to a susceptibility of extreme old age or to some idiosyncrasy.

Tests on Sheep

Tetrachlorethylene was administered to seven sheep (table VIII) in doses ranging from 5 cc to 20 cc after withholding food for 18 hours. The first five animals of this group were feeder lambs, which had been obtained from the stock-yards and selected because of their general unthriftiness and evidence of parasite infestation. The last two were aged ewes in advanced stages of pregnancy.

When the 5-cc globules of tetrachlorethylene were being administered to sheep 4, two were crushed between the teeth, allowing

the contents to escape into the mouth. No loss of the drug occurred, as it and the broken globules were almost immediately swallowed. Within a few minutes this animal showed a marked dizziness, falling on its side and lying in a stupor. These symptoms persisted more or less for about four hours, when the animal was again apparently normal. No further symptoms were noted until the second day when the animal refused all food. There was no return of appetite and the animal remained in a recumbent position during the greater part of the remaining days it was allowed to live. During the first two days the bowel movements were less than normal. There were none whatever during the last five days.

TABLE VIII—*Tetrachlorethylene to sheep in doses of 5 cc to 20 cc*

SHEEP	WEIGHT (LBS.)	C_2Cl_4 (CC)	WORMS PASSED	WORMS POSTMORTEM		
			STOMACH	TAPE	ROUND	WHIP
1	52.0	5.0	?	5	0	0
2	38.0	5.0	?	0	1	0
3	53.0	10.0	1	10	0	8
4	46.5	15.0	0	0	0	0
5	54.0	20.0	0	5	0	5
6	91.0	5.0	8	No autopsy		
7	81.0	5.0	0	No autopsy		

? Number undetermined.

On postmortem the liver was found to be about normal in color and size but rather soft and friable. The gall-bladder was filled with dark-green bile. These changes, no doubt due to the effects of the anthelmintic given, were probably more severe than they would have been had not the intussusception found in the colon been present. The resultant occlusion of the lumen of the intestinal tract undoubtedly had considerable influence on the absorption of the drug. While under observation, none of the other animals, either lambs or pregnant ewes, showed any visible effects of the drug. On postmortem, the internal organs of sheep 1, 2 and 3 were apparently normal, The liver of sheep 5 was light yellow in color, soft and friable. Thin, light-yellow bile filled the gall-bladder. No other changes were noted.

Sheep 6 and 7 were not autopsied, being held for further experiments.

All of these sheep showed more or less stomach-worm infestation, on preliminary fecal examination. While sheep 1 and 2 were known to have passed a considerable number of stomach worms, through an unfortunate chain of circumstances an accurate count could not be made. No stomach worms were found on postmortem of any of the animals, 5 cc of tetrachlorethylene being sufficient to remove all.

Apparently tetrachlorethylene has no effect on the tapeworms of sheep, since none was removed by any amount of the drug used. As in the dog, its effect on whipworms seems to depend on whether or not it comes in contact with these parasites.

Tests on Man

In addition to the effects previously noted by the author[3] and his assistant, after taking 1 cc each of tetrachlorethylene, the author on another occasion took 1 cc of the drug enclosed in a soluble elastic globule about three hours after a light lunch. No purgative was taken later. About one-half hour after taking the drug only a very slight sensation of dizziness was noted which persisted for about fifteen minutes. No later symptoms were noted.

Summary

Tetrachlorethylene given to chickens, dogs, cats, foxes, sheep, cattle and horses, in varying doses, caused no toxic symtoms except in a few cases, e. g., the chickens which received repeated large doses, horse 910 and sheep 4.

The different effects experienced by the author and his assistant on one occasion and by the author later, the symptoms shown by sheep 4 and horse 910 may have been due to some idiosyncrasy of the individuals somewhat similar to the tolerance for carbon tetrachlorid by certain foxes at certain times noted by Hanson and Van Volkenberg[7].

Tetrachlorethylene, like carbon tetrachlorid, is very effective against blood-sucking worms, as shown by its action on hookworms of dogs, cats, and foxes and on stomach worms of sheep. It also possesses a high efficacy against roundworms in dogs, cats, chickens and foxes.

Conclusions

The conclusions to be drawn from these experiments are as follows:

Tetrachlorethylene administered to dogs in soluble elastic globules, food being withheld until at least two hours later, is as effective as when given in hard gelatin capsules.

The administration of dry Epsom salts two hours after tetrachlorethylene does not lessen the efficacy of the anthelmintic in dogs. A slight reduction might occur if the salt is given an hour or less after the anthelmintic, enclosed in soluble elastic globules.

Tetrachlorethylene in doses up to 5 cc is not toxic to chickens. Larger doses given four days after that amount cause toxic symptoms. Further work with heavily infested birds is necessary to determine a minimum effective dose.

The dose rate for dogs is not excessive for cats. Tetrachlorethylene in these amounts is efficient against hookworms and roundworms of these animals, and can be given with safety.

In most instances 1 cc of tetrachlorethylene should prove effective against hookworms and roundworms of adult foxes. Only in exceptional cases, where the size of the animal warrants, would a dose of 2 cc be indicated.

Judging the effects noted following the use of tetrachlorethylene in dog puppies, no trouble should be anticipated following its use in fox puppies, although further experiments are necessary to determine its limitations in these animals.

Further trials are necessary to determine the effectiveness of tetrachlorethylene against hookworms and roundworms of cattle. The drug should prove as effective against these parasites of cattle as it does for the hookworm and roundworms of the dog and fox. The recommended dose of carbon tetrachlorid should prove efficient and non-toxic.

Administration of tetrachlorethylene to horses may produce transient symptoms of dizziness, unsteadiness, etc., possibly depending on the age and condition of the animal. Its efficacy against bots is probably slightly in excess of that of carbon tetrachlorid and less than that of carbon bisulphid.

Tetrachlorethylene, in doses of 5 cc, is effective against stomach worms of sheep. The drug can be given to feeder lambs and pregnant ewes without harmful reffects.

Tetrachlorethylene apparently has no anthelmintic action against tapeworms of any of the animals used in these experiments.

On account of its relatively high safety and its effectiveness against hookworms and roundworms of the animals used, tetrachlorethylene deserves further cautious use for the removal of these parasites from man.

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ARMY GRADUATION EXERCISES

The graduation exercises for the Army Medical, Veterinary and Dental schools of 1926 were held in the Red Cross House at the Army Medical Center, Washington, D. C., February 10, 1926, with appropriate ceremonies, including an address by the Surgeon General. The diplomas and medals were presented by Major General John L. Hines, Chief of Staff.

The following veterinary officers were graduated with honors: Captains George J. Rife (Hoskins Medal), Chauncey E. Cook, and and James E. Noonan.

The following members o the Veterinary Corps also were graduated: Captains Kenneth E. Buffin, Jesse D. Derrick, Sawyer A. Grover, Patrick H. Hudgins, and Howard M. Savage; 2nd Lieutenants Verne C. Hill, Harry R. Leighton, and Elmer W. Young.

The following officers of the Cuban Army also were graduated: Captain Rafael F. Santa Maria, and 1st Lieutenant Antonio M. Martínez.

ANTHRAX AGGRESSIN*

By C. E. SALSBERY, *Kansas City, Mo.*

Our knowledge of the variations in the pathogenicity of different species of bacteria leaves us in the dark on the question of virulence. The injection of massive doses of certain species of bacteria results merely in a temporary reaction, mostly local; while, in other cases, the injection of extremely small doses frequently causes fatal infection. The anthrax bacillus is a well-known member of the latter group.

Bail's aggressin theory was the result of his attempt to explain this phenomenon of virulence. In his experimental work with anthrax, he discovered that the serum of animals highly susceptible to anthrax often possessed distinct bactericidal powers against this bacillus. But when these animals were injected with doses much smaller than those their serum should surely destroy, they became fatally infected and succumbed rapidly, the bacilli being found in the blood in great numbers. The bactericidal power of the serum in vitro was ineffective in the body of the animal because here the bacilli were certainly defended by something that was not present outside of the body. This something must have had a definite effect upon their virulence.

He also demonstrated that the injection of sterilized exudates along with sub-lethal doses of bacteria produced fatal infection, whereas the injection of the bacteria alone had little effect. His theory therefore assumed that certain bacteria were virulent by virtue of definite substances secreted by them that overcame the defenses of the animal, permitted and even hastened the proliferation of these bacteria in the animal body. The primary effect of these substances was their power to inhibit phagocytosis. They were called aggressins and are present in the exudates about the point of inoculation in fatal infections.

The important and practical application of Bail's theory is that animals may be successfully immunized against these aggressins and that an animal so immunized is able to prevent infection, not because its serum is bactericidal but because the anti-agressins present neutralize the aggressive substances secreted by the bacteria, thus destroying their offensive powers and making them susceptible to phagocytosis. This immunity

*Presented at the third semi-annual meeting of the State Veterinary Medical Association of Texas, Forth Worth, Jan. 20-21, 1926.

may be passively transferred to other untreated animals. Animals treated with aggressins are actively immunized and the immune bodies in their sera are anti-aggressins.

The practical and highly successful application of aggressin therapy has been fully proven in the use of blackleg and hemorrhagic septicemia aggressins, and experimental data on the use of anthrax aggressin indicate that its practical application will be just as successful.

Pasteur's anthrax vaccines were highly successful in his own hands, but in their wholesale production and use they were not so successful and often defeated the purpose for which they were used by causing anthrax rather than preventing it. In districts where anthrax is extremely virulent, they failed to produce sufficient immunity in vaccinated animals to protect them against infection. Anti-anthrax serum and the higher spore vaccines have not prevented serious losses in these extremely virulent anthrax districts and the necessity of presenting animals for treatment more than once is an added disadvantage.

The development of anthrax aggressin therefore is the result of the successful use of aggressins in other diseases and the demand for an immunizing product against anthrax that could be used with much less inconvenience and expense and produce a more solid immunity without danger of producing the disease.

Our work was started in November, 1922, and has continued, with improvement in technic, so that now anthrax aggressin may be produced on a uniform schedule by a fixed process. The proper virulence of cultures required has been determined, the best method of inoculation and the most satisfactory amount and concentration of the culture that will kill the animals and produce a good exudate after the proper period of incubation have been determined, so that the animals die of anthrax with considerable regularity, producing a uniform quantity of exudate of the desired potency. To give in detail all of the problems encountered during this experimental work would require more time and space than the importance of the information would warrant.

The method of harvesting the exudate presented a serious problem at first and several methods were tried before we devised the one now employed. It is not a difficult problem nor is it attended by any particular risk to the operators. We remove all of the exudate from the dead animals without any loss. It is not exposed to contamination and persons working with it are

not in any more danger of becoming infected than if they were engaged in the ordinary laboratory work with anthrax. As soon as the harvesting is completed, the material is treated to kill the anthrax organisms in it. Clarification and final sterilization are accomplished by using high-speed centrifuges and Berkefeld filters.

The question of most importance is the one of potency. It is possible to produce aggressins of high potency or of low potency, depending upon the virulence of the cultures used, the amount and concentration of the inoculum and the length of time the animals live after injection. The variations in temperature, between summer and winter, influence the period of incubation and must be considered in making the injections. The condition of the animal appears to have little or no influence on the problem of production.

After the method of production was simplified and perfected, an extensive schedule of testing was planned in which a great many animals were used to demonstrate the following principles:

(1) Aggressive action
(2) Active immunity in susceptible animals.
(3) Anti-aggressins in immunized animals
(4) Passive transfer of this anti-aggressin immunity.

The planning and carrying out of this testing program was not as simple as it might at first appear. We encountered a variety of conditions that were not easily overcome and some of them will always prevail, simply because the individual factor in different animals can never be entirely controlled. In selecting animals for tests, we tried to get them as uniform in every respect as possible. The virus was always checked, so that it would kill susceptible animals in less than 100 hours. In preparing for an exposure, we inoculated susceptible animals in order to establish this time feature, and we then used the virus and dosage that killed the pre-test animals within the required limit. In spite of this care in the selection of the proper dosage of virus, we had unsatisfactory results, because in some instances where we used the same virus and dosage that killed our pre-test animals in seventy-two to eighty-two hours, our controls in the test did not die. In some instances, some controls died and others survived. In other instances, the control died after the one-hundred-hour period and while we consider such a test of value inasmuch as death was due to anthrax, yet we were better satisfied when death occurred within the limit we tried to fix.

In some of our earlier tests, we attempted to give the virus intravenously, in sheep, but found that these animals were extremely susceptible to intravenous injections of even the smallest doses of anthrax bacilli and that such severe exposure was not a fair test and in no way comparable with natural infection. Subcutaneous injections of virus were therefore used in sheep. In the tests where yearling cattle were used, the intravenous injection of virus was made because subcutaneous injections failed to kill with sufficient regularity in the required time. All test animals were properly isolated and carefully observed daily as to clinical conditions, such as appetite, general appearance, etc., and temperatures were recorded each morning and sometimes in the evening. No special attention was given to feeding except there was always a sufficient quantity of good feed and water accessible.

The tests to demonstrate aggressive action were not as extensively carried out as the tests for potency. The first of these tests was made in February, 1923, and six guinea pigs were used (see table I). Four of the pigs received aggressin as follows: 0.5

TABLE I—*Test to show aggressive action (February 21, 1923)*

GUINEA PIG	AGGRESSIN (CC)	SPORE VAC. NO. 1 (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	RESULTS
904	0.5	1.0	OK	OK	OK	OK	OK	OK	Lived*
905	1.0	1.0	OK	OK	OK	OK	Sick	Dead	Anthrax, 125 hrs.
906	2.0	1.0	OK	OK	OK	Sick	Dead		Anthrax, 110 hrs.
907	3.0	1.0	OK	OK	OK	Sick	Dead		Anthrax, 100 hrs.
908		1.0	OK	OK	OK	OK	OK	OK	Lived*
909		1.0	OK	OK	OK	OK	OK	OK	Lived*

*O. K. on eleventh day.

cc, 1 cc, 2 cc, and 3 cc respectively; two other pigs received no aggressin. All six pigs received 1 cc of spore vaccine No. 1, which is sub-lethal for normal guinea pigs. Pig 904, receiving 0.5 cc aggressin, remained alive, indicating the amount of aggressin was too small to make the quantity of virus injected lethal for that pig. The three remaining aggressin pigs died of anthrax and the ones receiving the larger doses of aggressin died in progressively shorter periods. The two control pigs receiving spore vaccine alone remained alive without any apparent effect from its injection.

Having shown that a sub-lethal dose of anthrax virus could be made to kill by the injection of aggressin, we were reminded of

Bail's statement that aggressin immunization might be a dangerous undertaking during an epizootic. We therefore inaugurated another test to show that any excessive aggressive action could be controlled by the simultaneous injection of a small amount of anti-aggressive serum. In this test eight guinea pigs were used (see table II). Two received 1 cc of aggressin, two received 2 cc and two received 3 cc each. Three of these pigs received 5 cc each of the serum in the same order; then each of the eight pigs received 1 cc of the spore vaccine No. 1. It will be seen that the small amount of serum injected into pigs 287, 288 and 289 counteracted the aggressive action, inasmuch as none of these pigs died of anthrax. Pig 288 died on the sixth day but anthrax could not be demonstrated. Control pig 290 died on the third day but we could find no evidence of anthrax.

TABLE II—*Test of aggressive action (July 2, 1924)*

GUINEA PIG	AGGRESSIN (CC)	SERUM (CC)	SPORE VAC. NO. 1 (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	RESULTS
284	1.0		1.0	OK	OK	Sick	Dead			Anthrax, 90 hrs.
285	2.0		1.0	OK	OK	Dead				Anthrax, 65 hrs.
286	3.0		1.0	OK	OK	Sick	Sick	Dead		Anthrax, 110 hrs.
287	1.0	5.0	1.0	OK	OK	OK	OK	OK	OK	Lived*
288	2.0	5.0	1.0	OK	OK	OK	OK	OK	Dead	Not anthrax
289	3.0	5.0	1.0	OK	OK	OK	OK	OK	OK	Lived*
290			1.0	OK	OK	Dead				Not Anthrax
291			1.0	OK	OK	OK	OK	OK	OK	Lived*

*O. K. on the tenth day.

There is possibly another condition in connection with aggressive action and that has to do with dosage. Too large doses of aggressin are possible, according to Bail. The animal body absorbs only a certain quantity and any excess remains as active aggressin for a certain length of time. Bail claims this excess may be a source of danger in animals exposed before sufficient anti-aggressins are produced to neutralize it. The experience with blackleg and hemorrhagic septicemia aggressins, where they have been used in proper dosage in infected herds, has not proven them to be dangerous and it is not probable that this negative phase in the use of anthrax aggressin is any more significant than in blackleg or hemorrhagic septicemia. However, we believe early vaccination should be practiced.

In testing anthrax aggressin for immunizing value, we used a large number of animals, including guinea pigs, rabbits, sheep,

cattle, horses and mules. In preparing our virus for exposure of the animals, we were careful in trying to make it kill within a reasonable length of time and not to overwhelm them with an unreasonable dose, because we believe that, under natural or field conditions, no animal is ever exposed to an overwhelming amount of the infective material. On the other hand, we observed that the artificial exposure of animals to anthrax could not be done with the same degree of certainty or regularity in all cases, regardless of the virulence of our cultures. We have noted that a dose of anthrax culture is not always infective to all of the susceptible animals to the same degree, in that some die quickly, others survive longer, and some do not die at all. This irregularity

TABLE III—*Potency test of anthrax aggressin against spore vaccine No. 2. Aggressin injected February 2, 1923; 0.5 cc spore vaccine No. 2, subcutaneously, March 7, 1923*

GUINEA PIG	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	RESULTS
890	0.5	OK	OK	OK	Lived*
891	0.5	OK	OK	OK	Lived*
892	0.5	OK	OK	OK	Lived*
893	1.0	OK	OK	OK	Lived*
894	1.0	OK	OK	OK	Lived*
895	1.0	OK	OK	OK	Lived*
896	1.5	OK	OK	OK	Lived*
897	1.5	OK	OK	Died	Not Anthrax, 72 hrs.
898	1.5	OK	OK	OK	Lived*
899	2.0	OK	OK	OK	Lived*
900	2.0	OK	OK	OK	Lived*
901	2.0	OK	OK	OK	Lived*
902	Control	Sick	Died		Anthrax, 50 hrs.
903	Control	Sick	Died		Anthrax, 50 hrs.

*O. K. on eighth day.

caused the repetition of a number of tests and was absolutely uncontrollable.

Table III represents a guinea pig test in which varying doses of aggressin were given and it shows that all of the pigs without exception were protected against a lethal dose of No. 2 spore vaccine, except the two controls, 902 and 903, which received no aggressin. The table shows that the killing period in these two controls was rather short; also that they died of anthrax. Pig 897 died on the seventy-second hour, but we were unable by any means to demonstrate anthrax.

Table IV represents a protective test against a culture of

anthrax bacilli, our culture 162, which was used after 24 hours of incubation in broth and the amount given each pig represented 5 m.l.d. The virus in this test was given subcutaneously and all of the pigs, except one which received 0.1 cc of aggressin and one

TABLE IV—*Potency test of anthrax aggressin against anthrax cultures. Aggressin injected March 12, 1923; 0.05 cc (5 m.l.d.) of anthrax culture 162 injected subcutaneously, March 22, 1923*

GUINEA PIG	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	RESULTS
934	0.1	OK	OK	OK	OK	OK	OK	Lived*
935	0.1	OK	OK	OK	OK	OK	OK	Lived*
936	0.1	OK	OK	OK	OK	Sick	Dead	Anthrax, 120 hrs.
937	0.2	OK	OK	OK	OK	OK	OK	Lived*
938	0.2	OK	OK	OK	OK	Sick	Dead	Anthrax, 120 hrs.
939	0.2	OK	OK	OK	OK	OK	OK	Lived*
940	0.5	OK	OK	OK	OK	OK	OK	Lived*
941	0.5	OK	OK	OK	OK	OK	OK	Lived*
942	0.5	OK	OK	OK	OK	OK	OK	Lived
943	1.0	OK	OK	OK	OK	OK	OK	Lived*
944	1.0	OK	OK	OK	OK	OK	OK	Lived*
945	1.0	OK	OK	OK	OK	OK	OK	Lived*
947	Control	Sick	Dead					Anthrax, 24 hrs.
948	Control	Sick	Dead					Anthrax, 36 hrs.

*O. K. on eighth day.

which received 0.2 cc of aggressin, survived the test and were well at its conclusion. It will be noted that the two controls, 947 and 948, died in a very short time of positive anthrax.

Table V shows the result of a guinea pig test against 24-hour, broth culture 172 and the amount given each pig represented 10

TABLE V—*Potency test of anthrax aggressin against 24-hour broth culture. Aggressin injected July 13, 1923; 10 m.l.d. of anthrax culture 172 injected subcutaneously, July 25, 1923*

GUINEA PIG	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	RESULTS
129	1.0	OK	OK	OK	OK	OK	OK	Lived*
130	1.0	OK	OK	OK	OK	OK	OK	Lived*
131	2.0	OK	OK	OK	OK	OK	OK	Lived*
133	2.0	OK	OK	OK	OK	OK	OK	Lived*
134	3.0	OK	OK	OK	OK	OK	OK	Lived*
135	3.0	OK	OK	OK	OK	OK	OK	Lived*
136	3.0	OK	OK	OK	OK	OK	OK	Lived*
140	Control	Dead						Anthrax, 18 hrs.
141	Control	Dead						Anthrax, 12 hrs.
142	Control	Dead						Anthrax, 18 hrs.

*O. K. on eighth day.

m.l.d. This culture was sufficiently virulent to kill the control pigs in eighteen, twelve and eighteen hours respectively.

Table VI shows the guinea pig test, using 24-hour, broth culture 172, which had been previously passed through four generations of guinea pigs and four generations of rabbits, for the purpose of exalting its virulence. Each animal received a dose representing 100 m.l.d. and we find that none of the animals were permanently protected against this excessive dose. The table shows that the two controls died first and the aggressin pigs show an interesting variation in protection, according to the amount of aggressin given. The controls died in 36 hours; pig 214, receiving 0.2 cc aggressin, lived 48 hours; pig 210, receiving 0.75 cc aggressin, lived 72 hours; pig 205, receiving 1 cc aggressin lived 100 hours. These hours are approximate, inasmuch as it is not always possible to be present just when the animal dies.

TABLE VI—*Potency test of anthrax aggressin against 24-hour broth culture. Aggressin injected August 29, 1923; 100 m.l.d. of anthrax culture 172 injected subcutaneously, September 11, 1923*

GUINEA PIG	AGGRESSIN (CC	1ST DAY	2ND DAY	3RD DAY	4TH DAY	RESULTS
214	0.2	Sick	Dead			Anthrax, 48 hrs.
212	0.5	Sick	Sick	Dead		Anthrax, 60 hrs
210	0.75	OK	Sick	Dead		Anthrax, 72 hrs.
205	1.0	OK	OK	Sick	Dead	Anthrax, 100 hrs.
217	Control	Sick	Dead			Anthrax, 36 hrs.
219	Control	Sick	Dead			Anthrax, 36 hrs.

Table VII shows the result of the test on calves, using varying doses of aggressin and the 24-hour, broth culture 172. It is noted that calf 11, receiving 2.5 cc aggressin, died of anthrax on the fourth day. Unfortunately calf 7 died on the third day, cause not determined. We were not able to demonstrate anthrax. Calf 12 survived the test in good condition, with no apparent effect at any time. Control 141 was sick on the second and third days and remained listless until the ninth day, when it died of anthrax. The test was repeated by using calves 8 and 9. These calves received the aggressin on the same date that calves 7, 11 and 12 did, and it was our intention to hold them several months for the purpose of testing the permanency of the immunity but we changed our program and they were used immediately following the conclusion of the first test. They received the same virus with the same dosage as the previous calves and the table shows that both calves 8 and 9 went through the test in excellent con-

TABLE VII—*Potency test of anthrax aggressin against culture No. 172, on yearling cattle. Aggressin injected August 14, 1923; 10 cc of culture 172 injected*

CALF	AGGRESSIN (CC)	CULTURE INJECTED	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	8TH DAY	9TH DAY	10TH DAY	RESULTS
11	2.5	11– 7–23	103.0	105.6	103.0	Dead							Anthrax
7	5.0	11– 7–23	102.7	105.8	Dead								Not Anthrax
12	7.5	11– 7–23	101.0	104.8	103.0	102.0	101.4	102.6	100.8	100.0	100.0	99.4	Lived
141	Control	11– 7–23	104.8	Sick 105.2	Sick 103.6	Sick 102.0	Sick 103.0	Sick 105.4	Sick 105.8	Sick 104.4	Dead		Anthrax
8	5.0	11–16–23	101.8	104.0	105.6	106.4	105.0	104.8	103.0	103.4	102.6	101.6	Lived
9	7.5	11–16–23	101.0	101.8	102.6	101.6	103.2	104.6	104.8	103.2	101.4	101.0	Lived
143	Control	11–16–23	101.2	105.0	106.0	104.4	Sick 106.0	Sick 105.4	Sick 104.4	Sick 104.2	104	Dead	Anthrax

TABLE VIII—*Anthrax aggressin test on calf. Aggressin injected January 28, 1924; 10 cc of anthrax culture (spore 172) injected June 28, 1924*

CALF	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	8TH DAY	9TH DAY	10TH DAY	11TH DAY	12TH DAY	13TH DAY	CONDITION
20	10.0	103.0	101.8	101.8	101.0	101.8	102.0	102.2	101.0	102.0	102.6	101.6	102.2	102.0	Alive
142	Control	101.8	101.6	104.8	Sick 102.6	Sick 102.4	Sick 102.6	Sick 102.2	Sick 103.6	103.4 a.m. Dead p.m.					Anthrax

dition, while calf 143 showed a high temperature on the second day and remained high until the ninth day, when it died of anthrax.

Table VIII represents a calf test, five months after the injection of anthrax aggressin. The virus used in this test was spore No. 172. This dose killed a large sheep in 72 hours from subcutaneous injection. Control was sick on the fourth day and each succeeding day, lying down most of the time with frequent groans and did not eat. Constipation was evident. Blood smears after death revealed large numbers of anthrax organisms, which were also recovered in cultures. This test indicated that the aggressin immunity has a degree of permanency.

Table IX shows a test in which the virus was given subcutaneously to sheep. Before inaugurating this test we prepared a virulent spore suspension of culture 172 and determined the killing dose by using several animals and the dose which we used killed our experimental m.l.d. sheep in 85 hours, of anthrax. In this test, we used aggressin alone, aggressin with serum, and serum alone. The table shows that the three animals receiving 5 cc of aggressin survived and none of them had a very high temperature at any time. Sheep 45, receiving 10 cc of aggressin, died on the 140th hour, of anthrax. Two sheep, 87 and 54, receiving 10 cc of aggressin and 10 cc of serum, simultaneously, remained well, while sheep 61 and 84, receiving serum alone, died of anthrax. Our control showed the individual factor to which we called attention elsewhere in this report, in that it survived the dose of virus for 188 hours, approximately 100 hours longer than our experimental m.l.d. sheep, which died in 85 hours.

Table X shows a potency test in which six sheep received varying doses of aggressin, with two controls receiving virus only. Our pre-test dose killed the m.l.d. sheep in 72 hours. It will be noted in the table that controls 282 and 283 responded to the virus in practically the same time that our pre-test sheep did. The potency of the aggressin is well shown, in that it protected perfectly in doses as small as 0.5 cc. Some high temperatures developed during the latter part of the test, but in all of these animals it was due to keratitis. Sheep 264 became totally blind; sheep 263 was partially blind. In spite of any devitalizing effect this may have had on the animals, they remained alive and were well, 30 days after the injection of the virus.

Table XI shows a potency test the result of which was quite satisfactory. Out of eight treated animals, two died of anthrax

TABLE IX—*Anthrax aggressin and serum test. Aggressin and serum injected March 8, 1924; 10 cc of anthrax culture (spore 172) injected April 7, 1924*

SHEEP	AGGRESSIN (CC)	SERUM (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	8TH DAY	9TH DAY	10TH DAY	11TH DAY	12TH DAY	CONDITION
80	5.0		104.2	101.0	102.4	102.8	103.4	103.4	102.2	103.0	102.0	101.0	101.0	101.0	Alive—Well
60	5.0		103.6	103.4	104.0	104.2	103.8	102.6	103.0	102.4	103.0	101.6	102.0	102.6	Alive —Well
71	5.0		104.8	101.4	103.2	103.4	103.4	101.2	104.0	103.4	102.4	101.0	102.0	101.6	Alive—Well
98	10.0		104.4	103.4	102.2	103.0	104.0	102.4	103.6	103.8	103.2	102.4	103.0	102.2	Alive—Well
45	10.0		102.8	102.6	102.2	105.8	107.0	107.4	Dead						Anthrax
87	10.0	10.0	102.4	101.8	103.0	103.4	104.8	106.2	106.0	103.0	104.0	103.6	103.6	102.0	Alive—Well
54	10.0	10.0	103.6	103.2	104.0	104.0	103.8	102.6	103.0	102.8	103.0	101.4	102.6	100.2	Alive—Well
61		10.0	103.4	103.6	102.8	101.8	106.0	105.0	107.8	Dead					Anthrax
84		10.0	103.6	104.0	104.2	104.2	104.2	104.8	105.4	104.8	Sick 106.8	Sick 108.0	105.4	Dead	Anthrax
Virus Control			102.4	102.0	102.2	102.4	104.0	102.4	Sick 104.2	Sick 106.2	Dead				Anthrax

TABLE X—*Anthrax aggressin test. Aggressin injected May 24, 1924; 10 cc of anthrax culture injected June 27, 1924*

SHEEP	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	8TH DAY	9TH DAY	10TH DAY	11TH DAY	12TH DAY	13TH DAY	14TH DAY	15TH DAY	16TH DAY	17TH DAY	18TH DAY	19TH DAY	20TH DAY	21ST DAY	RESULTS
263	0.5	105.0	104.0	103.4	103.6	103.4	102.6	103.8	104.4	104.6	103.6	106.0	104.4	104.8	103.4	104.4	105.2	104.4	102.4	103.0	102.8	102.6	Lived
264	0.5	104.8	101.4	103.0	103.6	102.8	103.0	104.0	104.0	104.2	103.8	103.8	104.0	103.4	102.2	103.8	103.6	104.2	104.2	105.6	103.0	104.0	Lived
265	1.0	102.8	103.6	102.4	102.6	103.0	102.0	104.0	103.0	104.4	103.0	104.0	104.2	102.6	103.0	103.2	102.6	102.8	102.8	102.0	102.2	102.8	Lived
266	1.0	104.0	102.0	102.4	103.0	103.2	102.8	103.4	103.6	103.0	104.0	104.0	103.2	103.0	102.8	103.4	103.0	103.0	103.6	103.8	103.6	103.2	Lived
267	3.0	105.2	103.4	102.8	102.0	102.0	102.0	103.4	106.6	104.6	103.4	106.2	106.6	104.2	104.4	104.0	104.2	103.6	102.8	103.6	103.6	103.0	Lived
268	3.0	103.8	103.0	103.0	103.0	102.8	102.4	103.6	103.0	103.6	103.4	104.6	105.4	104.4	102.8	103.0	102.6	103.0	103.0	103.6	103.0	102.8	Lived
282	Control	103.0	102.0	Sick 103.2	Sick 103.4	Dead																	Anthrax 100 hrs.
283	Control	103.2	105.4	Sick 103.6	Dead																		Anthrax 80 hrs.

TABLE XI—*Anthrax aggressin potency test on sheep. Aggressin injected September 5, 1924; 10 cc of anthrax culture (spore 172) injected September 27, 1924*

SHEEP	AGGRESSIN (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	8TH DAY	9TH DAY	10TH DAY	11TH DAY	12TH DAY	CONDITION 10–11–24
1	0.5	104.4	103.6	102.8	102.8	102.6	102.0	102.0	102.0	102.4	102.2	103.0		Alive
2	0.5	103.0	101.2	102.8	102.0	Sick 102.4	Sick 103.4	Sick 105.0	105.0	Sick 103.6	Sick 103.6	Sick 103.6		Alive
3	1.0	101.8	103.2	102.0	101.6	102.4	102.4	102.0	102.2	101.8	102.6			Alive
4	1.0	102.0	102.6	102.4	102.0	101.8	101.6	101.6	Sick 105.2	Dead 8 a.m.				Anthrax
5	3.0	103.4	103.4	102.6	102.8	103.8	103.0	102.8	102.6	103.0	103.8	Sick 102.6	Dead	Anthrax
6	3.0	103.0	102.2	102.2	102.0	102.0	104.4	102.0	103.0	104.0	103.0	102.4		Alive
7	5.0	102.4	102.8	102.8	Sick 102.2	Sick 104.0	Sick 104.0	103.8	101.8	101.6	102.6	102.0		Alive
8	5.0	102.2	101.8	102.0	101.4	102.0	102.0	102.8	102.0	101.6	102.4	103.2		Alive
9	Control	102.8	101.8	101.8	Sick 102.8	Sick 103.2	Sick 103.0	Sick 106.6	Dead 8 a.m.					Anthrax
10	Control	102.6	102.0	102.4	103.8	Sick 102.2	Sick 102.6	Sick 105.0	Sick 105.4	Sick 105.8	Dead 12 m.			Anthrax

and six survived. The controls died late, but anthrax was the cause of death.

Having concluded the schedule of tests shown above and a number of others, we decided to test our aggressin in another manner. For this work, we purchased an aged mare, in poor physical condition, and injected 10 cc of anthrax aggressin subcutaneously, on August 9, 1924. On August 26, 1924, she received, intravenously, 1 cc of a 24-hour virulent anthrax broth culture. Increasing injections were continued at seven-day intervals, until on Oct. 1, 1924, she received intravenously 75 cc of a 24-hour virulent culture, containing approximately 450 billion germs. No severe reactions were noticed as a result of the injections and, at the end of the injection period, the mare had improved in her physical condition and had gained considerably in weight. On Oct. 11, 1924, she was bled in the regular way and was carried on regular bleeding-and-injection schedule. This is a remarkable condition. In preparing animals for anti-anthrax serum production, weeks of preliminary treatment are necessary. The treatment begins with serum and spore vaccine, then special spore vaccine, followed by very small doses of living culture and increasing slowly until the 1-cc injection is reached. Ordinarily this requires a period of about eight weeks. In this experiment, the 10-cc aggressin injection produced as much immunity as the old method produced in eight weeks.

Believing that we could shorten the period of hyperimmunization still more, we purchased an aged mule, known as No. 75, and on Sept. 25, 1924, we injected 7 cc of aggressin subcutaneously. On Oct. 11, 1924, we injected intravenously 5 cc of a 24-hour virulent anthrax culture, containing approximately 30 billion germs, causing a severe temperature reaction and some apparent physical distress. Simultaneously with the exposure of mule 75, another mule (C-1) and one horse (C-2) were exposed in the same manner as controls. Mule C-1 was dead in 12 hours. This is very unusual for anthrax and the direct cause of death was probably due to shock or embolism, notwithstanding the presence of large numbers of anthrax bacilli in the blood. Horse C-2 was dead in 72 hours of anthrax, the bacilli being found in the blood. The second injection of 10 cc was made on Oct. 18, 1924, to mule 75 and on Oct. 25, 1924, a 20-cc intravenous injection was given and the injections were continued on the same schedule as our regular anthrax serum animals and the mule became one of our regular producers.

On Nov. 18, 1924, two mules (130 and 133) were each given 10 cc of aggressin subcutaneously. On Dec. 4, mule 130 received 5 cc of a 24-hour broth culture intravenously. One control animal received the same amount of culture in the same way. On Dec. 16, mule 130 received 10 cc of a 24-hour broth culture and continued on regular schedule and is still producing anthrax serum after more than one year. The control died of anthrax on the fifth day. Mule 133 received the first exposure on Dec. 12, and was carried on the same schedule as mule 130. This animal is also producing anthrax serum after more than a year on treatment and bleeding. The records of these animals are a part of our daily regular anthrax serum production schedule. This is a remarkable achievement and gives further evidence

TABLE XII—*Potency test on anti-aggressin serum. Anti-aggressin serum injected October 4, 1924; 0.5 cc broth culture (172) injected October 11, 1924*

RABBIT	SERUM (CC	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	CONDITION 10-27-24
709	1.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
710	1.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
711	2.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
712	2.0	OK	OK	OK	OK	OK	Sick*	Dead	Not anthrax
713	3.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
714	3.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
715	4.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
716	4.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
717	5.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
718	5.0	OK	OK	OK	OK	OK	OK	OK	Alive—Well
C-1	Control	OK	OK	OK	Sick	Dead a.m.			Anthrax
C-2	Control	OK	OK	OK	Dead				Anthrax

*Ear infected and badly swollen.

that it is possible to produce active immunity against anthrax by the injection of anthrax aggressin.

To demonstrate the possibility of producing anti-aggressin serum, we purchased two horses and on Aug. 21, 1924, each received subcutaneously 25 cc of anthrax aggressin. On Aug. 28, 1924, each received 50 cc. Then each received 100 cc of aggressin subcutaneously, at seven-day intervals, until five such injections had been given. Test bleedings were taken and twelve rabbits were used in one test (see table XII). Two received 1 cc each of the anti-aggressin serum, two received 2 cc each, two received 3 cc each, two received 4 cc each, and two received 5 cc each. Two controls received none. Seven days

later, each of the 12 rabbits received 0.5 cc of 24-hour, broth culture 172. The two controls were dead of anthrax on the morning of the fifth day, while all of the vaccinated rabbits, except 712, remained well. This rabbit died of ear infection. Swelling was noticed on the second day and the condition developed as the usual infectious otitis common in rabbits.

Another test was made, giving the anti-aggressin serum and virus simultaneously (see table XIII). This table is self-explanatory and requires no comment except that anthrax bacilli were demonstrated in the blood of both controls, 725 and 726.

While the testing of the anti-aggressin serum was not as extensive as the tests on aggressin, we believe the evidence produced in these two tests indicates that these two horses produced

TABLE XIII—*Potency test on anti-aggressin serum, October 16, 1924. Dose of anthrax cultures (162 and 173), 0.5 cc.*

RABBIT	SERUM (CC)	1ST DAY	2ND DAY	3RD DAY	4TH DAY	5TH DAY	6TH DAY	7TH DAY	CONDITION
719	1.0	OK	OK	OK	OK	OK	OK	OK	Alive*
720	1.0	OK	OK	OK	OK	OK	OK	OK	Alive*
721	3.0	OK	OK	OK	OK	OK	OK	OK	Alive*
722	3.0	OK	OK	OK	OK	OK	OK	OK	Alive*
723	5.0	OK	OK	OK	OK	OK	OK	OK	Alive*
724	10.0	OK	OK	OK	OK	OK	OK	OK	Alive*
725	Control	OK	Sick	Dead					Anthrax
726	Control	OK	Sick	Dead					Anthrax

*O. K. on eleventh day.

a highly potent anti-aggressive serum and that we succeeded in transferring this immunity passively to other animals.

After concluding such an extensive program of testing, we were convinced that anthrax aggressin possessed sufficient virtue to recommend its use against natural infection; in fact, we felt that it should be given a thorough field test before a definite statement could be made regarding its actual value under natural conditions. We believe in d'Herelle's opinion that the study of immunity in artificially produced infectious diseases does not necessarily mean that the same conditions will prevail under natural infection. Therefore, early in 1925, preparations were made to use anthrax aggressin in some of our southern states where anthrax occurs in very virulent form and where other methods of immunization have frequently failed to give proper protection.

Arrangements were made in a most careful manner and a thoroughly competent veterinarian, familiar with southern anthrax conditions, was detailed to use aggressin personally and only in districts where the disease occurred in its most virulent form. Districts in Mississippi, Louisiana, Arkansas and southern Texas were chosen and he was instructed to use it only on premises where there were heavy losses the previous year. Each treated animal was to be indicated by a permanent ear-tag and a sufficient number of animals left untreated to serve as controls. Arrangements with the local veterinarians were to be made so that they could be present when the aggressin was administered, keep in touch with the animals during the season, make examinations of any that were reported dead by the owners, hold autopsies and, when possible, confirm diagnosis by laboratory examinations of specimens. The plan worked exceedingly well and the local veterinarians and owners gave their full cooperation in making the tests reliable and of real value. In all, 12,685 animals, mostly cattle, were treated without any expense to the owners. Unfortunately anthrax did not break out except in some of the districts in southern Texas, but fortunately a large number of animals had been treated in these districts. The results of our field test, may be summarized as follows:

In Mississippi and Louisiana, where anthrax did not appear in 1925, 1,759 animals were aggressin-treated.

In southern Texas, where anthrax appeared in virulent form, 10,814 animals were aggressin-treated, 2,684 animals were left untreated and exposed, in the same pastures with the treated animals. Among the vaccinated animals, 35 deaths from anthrax occurred, representing a loss of .32 of 1%. Seven hundred twenty-six deaths from anthrax occurred among the unvaccinated exposed animals, representing a loss of 27%. One hundred sixty-three deaths occurred in these herds, before vaccination.

On one badly infected plantation in Arkansas, where anthrax has been extremely virulent, 77 mules were given the triple treatment with anthrax serum and spore vaccines. One hundred twelve cattle were given 5 cc of aggressin each and ear-tagged, and 100 cattle were left untreated. There were also 85 sheep left untreated. During the season, 11 of the 77 mules died of anthrax. Five of the 112 aggressin-treated cattle and 60 of the 100 untreated cattle died of anthrax. Of the 85 sheep, all but one died of anthrax. There were approximately 14% losses in the mules; approximately 4.4% in the aggressin-treated cattle;

60% in the untreated cattle and approximately 99% in the untreated sheep.

Conclusions

From the results of our work, we believe that aggressin immunization against anthrax offers distinct advantages.

We have demonstrated aggressive action.

We have shown that it produces active immunity.

We have shown that anti-aggressive serum may be produced by the gradual immunization with aggressins and that this anti-aggressive immunity may be passively transferred to other animals.

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FUR FARMS IN U. S. VALUED AT $18,000,000

Fur farming in the United States and in Alaska is steadily developing, says the Biological Survey of the Department of Agriculture, and certain phases of the industry are becoming a permanent addition to agricultural production. For the year 1924, there were approximately 2000 fur farmers in the United States and Alaska, the majority of whom were raising silver and blue foxes. The total investment in the business is somewhere between $15,000,000 and $18,000,000.

MEDICAL STUDENT STATISTICS

There were 18,200 medical students enrolled in the 80 medical colleges of this country last year, as against 12,930 in 1919. This is an increase of almost a thousand students per year during this six-year period. The number of colleges has remained substantially the same. From these colleges, there were 3974 graduates during the year which ended June 30, 1925. This was the largest number since 1913. Seventy-one colleges are rated as Class A, three as Class B, and six as Class C. The average number of students in the Class B and C schools was 78, while the average enrollment in the Class A schools was 246. Jefferson Medical College had the largest enrollment, with 573 students; Michigan, 533; Rush, 531; Harvard, 506; Tufts, 476; Pennsylvania, 471.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

ACUTE DILATATION OF THE HEART AND PHARYNGEAL PARALYSIS

By GREGORIO SAN AGUSTIN

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University of the Philippines
Los Banos, Laguna, P. I.

This case was assigned to Mr. Eleuterio Galope, senior student of the College.

On August 3, 1925, a hard-working pony, 8 years old, was brought by the owner to the hospital of the College, with the complaint that the animal was not eating well.

The animal was treated for angular mouth. The teeth were floated, and a tonic mixture was subsequently administered. Shortly after, the case returned with the following symptoms: pulse, 80; respiration, 42; temperature, 42.3° C. Appetite was impaired, and strong peristalsis was heard over the region of the small intestines. Dry, wheezing respiratory sound was also heard upon auscultation. Microscopic examination for blood disease was negative. The ailment was provisionally diagnosed as mycotic enteritis.

Treatment was instituted accordingly, consisting of quinin sulphate in a capsule to combat the high fever. The following day, he was given a purgative of aloin. White liniment was applied over the thorax. On the third day, a tonic mixture was administered, and the application of white liniment was continued. The animal greatly improved, and was discharged on the fourth day.

Two weeks later, the animal was brought again to the hospital, with the history that he was coughing at irregular intervals, appetite good and, the evening before, the animal was subjected to heavy work.

Clinical examination: Animal was in a serious condition; ventral part of the head greatly swollen; pulse, 48; respiration, 12; temperature, 38.8° C. Weakness of the heart was noticed;

absence of vesicular sound on the right lobe of the lung and hardly audible on the left lobe; muco-purulent nasal discharge, and a very pronounced dysphagia, feed and water regurgitating from the nostrils. The extremities were cold, and the visible mucous membranes were slightly icteric.

Urine analysis: Specific gravity, 1030; acid in reaction and contained slight mucin; albumen test was negative; bile pigments present.

Blood examination: Stained smears of blood showed streptococci; erythrocytes appeared vacuolated, which was an indication of anemia. Bronchial cells were also present. Blood examination for infectious disease was found negative.

The heart was strongly palpitating, which did not correspond to the weak pulse. The heart region was greatly increased. The heart-beat could be felt as far back as the eighth intercostal space. Systole was very much prolonged.

It was then diagnosed as acute dilatation of the heart and pharyngeal paralysis.

Treatment: The line of treatment was almost purely symptomatic, and consisted of cardiac tonics and stimulants, diuretics, medicinal inhalations, rubefacients, digestive stimulants and local applications of antiphlogistin over the pharynx. In spite of all the treatment instituted, besides good care and attention, there had been no sign of improvement.

Daily symptoms: During the course of the disease, temperature varied from 38.4° C. to as high as 42.3° C.; pulse, from 44 to 120, and respiration from 10 to 42 per minute, frequently abdominal in type. Appetite had not been so greatly impaired, as the animal showed great desire to eat, but the greater part of the food had been constantly regurgitating through the nostrils, due to the pronounced dysphagia. The nasal discharge was muco-purulent and, at the later part of the disease, it became purulent. Cough occurred at certain intervals, and emaciation gradually took place. Heart weakness was marked, mucous membrane of the conjunctiva petechiated, and consciousness partly impaired. The pharyngeal affection did not show any improvement during the whole course of the disease, so that this was doubtless an influencing factor in causing the foreign-body pneumonia.

Course: After an illness of 36 days, without any appreciable improvement and a gradually progressive weakness, the animal died with marked emaciation.

Autopsy: There were no external manifestations except a bloody, fetid discharge from the nose. Nasal, pharyngeal, laryngeal and tracheal mucosae were markedly congested; anterior halves of both lobes of the lung were hepatized and gangrenous, which was attributed to the pronounced dysphagia; evidently the food must have entered the respiratory tract and produced the mechanical pneumonia. Heart was slightly hypertrophied, but the right chambers were severely dilated. Heart muscle, liver and spleen showed cloudy swelling; kidneys, fatty degeneration. The pleura was inflamed and showed deposits of fibrin on its visceral surface. The pericardial sac was distended with bloody fluid.

OHIO CONFERENCE

The College of Veterinary Medicine, Ohio State University, announces a veterinary conference for graduate veterinarians, to be given March 24-26, 1926.

The program, arranged under the direction of Acting Dean O. V. Brumley, is one the veterinarians should appreciate. The scope and character of the contributions and the high standing of the contributors in their respective places, presages a conference of more than usual significance to the veterinarians of this state, and holds out the prospect of a large attendance.

Dr. John W. Adams, president of the American Veterinary Medical Association, will speak on both professional questions and surgery; Dr. L. Van Es, of the University of Nebraska, will lecture on poultry diseases; Dr. M. Dorset, of the U. S. Bureau of Animal Industry, will discuss "Hog Cholera Control" and Dr. Howard J. Milks, of Cornell University, will give a lecture and demonstration on "Digestive Disorders of the Dog."

The local contributors include the faculty of the College of Veterinary Medicine and the following: G. W. Rightmire, Acting President of Ohio State University; Carl W. Gay, Professor of Animal Husbandry, Ohio State University; E. L. Dakan, Head of Poultry Husbandry, Ohio State University; James M. Phillips, Pasteur Institute, Columbus, Ohio; John Lyman, Head of the Department of Agricultural Chemistry, Ohio State University.

The Military Director has dissolved the Spanish National Veterinary Association, at Madrid, because it was considered to be a disturber of public peace.

REVIEW

OFFICIAL HISTORY OF THE WAR VETERINARY SERVICE. By Major-General Sir L. J. Blenkinsop, K. C. B., D. S. O., and Lieutenant-Colonel J. W. Rainey, C. B. E. 782 pages. His Majesty's Stationery Office, London, England, 1925. Cloth, 21sh.

This book is a well-written history of 782 pages, giving a detailed account of the organization and activities of the Royal Army Veterinary Corps in the World War. It contains a list of charts, plans, diagrams, photographs, thirty-one chapters in the text and a complete list of service announcements and pamphlets. It may be considered a text-book as well as a history. It shows the importnace of having a plan of organization ready to put into operation.

Our British confreres profited by experience in the Boer War; their service at that time was unorganized. During the interim between then and the World War they planned and adopted an organization that was equal to any emergency. It was followed and improved upon during the War. Its usefulness and worth were proven beyond all doubt. A grand total of 2,562,549 incapacitated animals passed through their service; seventy-eight per cent of this number were cured and returned to duty. They had at times as high as 110,000 patients daily under treatment. The list of diseases and injuries to horses, mules, and camels covered nearly everything known from the tropics of Africa to Northern Russia.

The History shows the enormous task to be done to control waste and inefficiency in public animals during war. It describes fully and lucidly how the Royal Veterinary Corps was organized, trained, and did the work. There is no indication of bragging or boasting. Mistakes and bad management are recorded. Blame is placed where it belongs.

The Corps was dependent upon no other branch of the War Department. It had its job and did it to the satisfaction of all concerned. All animals were under the kind and capable protection of the Corps from the time of purchase. Sick or injured animals were promptly placed under proper care and treatment and were neither neglected nor permitted to be worked until conditions were hopeless and incurable. A military necessity

has often been used unnecessarily as an excuse for overwork and neglect in care and feeding; these faults were reduced to a minimum through the watchfulness and efficiency of the Veterinary Corps. This was the first time in which glanders was successfully controlled during war and not spread by public animals after hostilities had ceased. The same is true of practically all other communicable diseases. The History shows fully how these diseases were controlled. Comparatively few drugs were used. The chief effort was devoted to preventive measures. This was practiced faithfully and efficiently from the time an animal was purchased. The excessive waste in animals could be checked in civil life by an application of their regulations for shipping horses by train or boat. Many valuable suggestions are given for treating wounds, injuries, and the ordinary diseases that are common in civil life.

One is disappointed, at first, in glancing over the outline of the History at the inconspicuous part devoted to the service in France. This disappointment is dispersed after a perusal of the book; the service in France was only one unit in the many theaters of operation; the service was conducted on practically the same plan in each one. It was the aim of the Corps to do the work wherever duty called. Those who are expecially interested in the British Service in France should also read "Army Veterinary Service in War," by Major-General Sir John Moore.

The work of the Royal Veterinary Corps was the ideal of all veterinary officers of the Allied Nations. The service was greatly appreciated by those interested in animals from an economic as well as from a humane standpoint. We are fortunate in having its activities so well recorded. Our congratulations are extended to those who planned the organization, authorized its adoption, put it into successful operation, and also to those who compiled the History.

C. J. M.

ABSTRACTS

CHEMOTHERAPEUTIC STUDIES IN RABIES. Malcolm J. Harkins. Jour. Amer. Med. Asso., 84 (1925), pp. 1797-1798.

The studies reported were conceived with the idea that possibly the use of some of the more recently used chemotherapeutic compounds might have an effect on the virus of rabies and also

divulge a lead to the successful treatment of other diseases, the etiology of which is classed as an ultramicroscopic virus.

The experiments were conducted on rabbits, fixed virus (rabbit medulla) being used for infection. All injections were made intracranially. The emulsion for infection was prepared by suspending a piece of medulla 0.5 cm. long in 4 cc of sterile 0.85% sodium chlorid solution. This was used in most cases in doses of 0.2 cc.

No germicidal effect was exerted within thirty minutes at 38° C., as evidenced by subdural tests on rabbits, when the fixed rabbit virus was suspended in a 1:200 dilution of sulpharsphenamin, 1:500 and 1:1000 dilution of flumerin, and a 1:1000 dilution of sodium-oxymercury-orthonitrophenolate.

No effect was observed following the injection of gentian violet, neo-arsphenamin, sulph-arsphenamin, potassium bismuth tartrate, ethylhydrocuprein hydrochlorid, quinin and urea hydrochlorid or mercurochrome—220 soluble, either preventive or curative. No prolongation of the incubation period was noted even when some of the compounds were injected as early as from two to four hours after infection and continued daily until death.

S. S.

THE DOG AS A CARRIER OF DISEASE TO MAN. Nunas Joannides and William A. Riley. Jour. Amer. Med. Asso., 85 (1925), p. 1054.

No attempt is made to give a detailed discussion of the various diseases that a dog may carry.

There are two modes of disease transmission by the dog; first, when the dog contracts a disease and carries it to human beings or when it acts as an intermediate host of various parasites; second, by coming in contact with some infected article and carrying the infection into the household.

In the presence of infectious diseases, the dog should be quarantined with the rest of the household. Periodically vermifuges should be given, this treatment as well as those for the various skin diseases being best carried out by the veterinarian.

Epidemics of rabies should be a danger signal and an indication to muzzle all dogs in the neighboring localities.

S. S.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Captain Howard M. Savage relieved from duty at the Army Veterinary School and from temporary duty at Carlisle Barracks, Pennsylvania, effective on completion of course of instruction at the Medical Field Service School on or about June 4, 1926, and will then report to the Commanding Officer at Plattsburg Barracks, N. Y., for duty.

Reserve Corps

New Acceptances

First Lieutenant:
Ikard, Elijah Harrison.....Gooding, Idaho (transferred from Cav-Res).
Second Lieutenant:
Robertson, Willard Thomas..Hale, Mo.

Promotions

Major Ora Ples Davis, Box 241, Lewiston, Mont., promoted to grade of Lieutenant Colonel.

First Lieut. Horace Leslie Anderson, Rockwell, Iowa, promoted to grade of Captain.

First Lieut. Edwin Frank Kallenberg, 138 So. 25th St., East Cedar Rapids, Iowa, promoted to grade of Captain.

First Lieut. James Alex. McKitterick, Jr., Greenwood, Mo., promoted to grade of Captain.

Second Lieut. Leroy Emery Dietrich, 828 Porter St., Wichita, Kans., promoted to grade of First Lietuenant.

Second Lieut. Ralph Edgar Simonsen, Marcus, Iowa, promoted to grade of First Lieutenant.

Separations

First Lieut. Adolph H. Legenhausen, 111 Federal Bldg., Des Moines, Iowa, failed to accept reappointment.

First Lieut. James Robert Renfrew, 1714 Locust St., Kansas City, Mo., failed to accept reappointment.

Second Lieut. Michael James McGinty, 829 Washington St., Indianapolis, Ind., declined reappointment.

HORSES FOR FOOD GO TO EUROPE FROM CANADA

Reports from Saskatchewan, Canada, state that 50,000 Northwest range horses are being bought for shipment to Europe, principally Belgium, Spain and France. There they will be slaughtered and the dressed meat turned into human consuming channels. In Northwest Canada the reported price is $3 a head and, as the syndicate is said to be taking all offerings, ranchmen are eager to accept the price for the old and less useful kinds.

The New York Times Magazine says "the donkey is passing." Now and then he can be seen passing with one hand on the wheel and his other arm around a girl.

MISCELLANEOUS

MEMORIAL TO DR. RANSOM PROPOSED

A committee consisting of Drs. M. C. Hall and E. B. Cram, of the Department of Agriculture; C. W. Stiles, of the Public Health Service; H. J. Nichols, of the Army Medical Corps, and W. W. Cort, of Johns Hopkins University, has been considering a proposal to establish a memorial to the late Dr. Brayton H. Ransom who, at the time of his death, was chief of the Zoological Division of the Bureau of Animal Industry, and who ranked as one of the foremost parasitologists of the world. The several propositions which are being considered as to the form of the memorial are as follows:

A tablet or painting, to be put up in the Department of Agriculture; a medal or a money prize, to be awarded for important contributions in medical zoology, or a scholarship, to provide for carrying out further investigations of the sort to which he devoted his life. The final decision will depend on the amount subscribed. The subscriptions may be sent to the secretary of the committee, Dr. Eloise B. Cram, Bureau of Animal Industry, Department of Agriculture, together with suggestions as to the nature of the memorial.

This proposed memorial is being brought to the attention of parasitologists, zoologists and physicians, in addition to veterinarians. It is well known to veterinarians that Dr. Ransom's work was in the field of veterinary parasitology and that in that field he made many important contributions to veterinary science. The routine procedures of the meat inspection service of the Bureau of Animal Industry in dealing with parasitic conditions are based on his investigations, alone and in collaboration with his associates. The dipping procedures of the field inspection service are based on his investigations and those of his associates. Most of our knowledge in regard to parasites present in this country and in regard to their life-histories is due to him. As a result of his life history studies he has developed some of our most serviceable prophylactic measures, including the extraordinarily successful swine sanitation system which is now spreading throughout the Corn Belt from McLean County, Illinois. Nor were the everyday problems of the practitioner neglected; present-day anthelmintic medication in the treatment

of worm infestations is based on the work carried out in Dr. Ransom's division under a project initiated by him in 1915.

The veterinarians of North America are familiar with Dr. Ransom and his work. Many of them knew him as a personal friend. He was an honorary member of the American Veterinary Medical Association and keenly interested in the veterinarian's problems. Undoubtedly many of them will wish to contribute to a memorial in his honor.

CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces the following open competitive examination:

Junior Veterinarian

Receipt of applications for junior veterinarian will close March 27. The date for assembling of competitors will be stated on the admission cards sent applicants after the close of receipt of applications.

The examination is to fill vacancies in the Bureau of Animal Industry, Department of Agriculture, for duty in the field, and in positions requiring similar qualifications.

The entrance salary is $1,860 a year. After the probational period required by the civil service act and rules, advancement in pay may be made without change in assignment up to $2,400 a year. Promotion to higher grades may be made in accordance with the civil service rules as vacancies occur.

Applicants must have been graduated from a veterinary college of recognized standing, or must be senior students in such an institution, and furnish proof of graduation during the existence of the eligible register resulting from this examination; provided, that the names of senior students who pass the examination will not be certified for appointment until they have furnished proof of actual graduation.

Competitors will be rated on veterinary anatomy and physiology, veterinary pathology and meat inspection, and theory and practice of veterinary medicine.

Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., or the secretary of the board of U. S. Civil service examiners at the postoffice or customhouse, any city.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Proceedings of Sixty-second Annual Meeting, Portland, Oregon, July 21 to 24, 1925

SECTION ON GENERAL PRACTICE

WEDNESDAY MORNING, July 22, 1925

The meeting was called to order at 9:30 a. m., by Dr. E. R. Steel, chairman.

Dr. Steel delivered his address.

GENTLEMEN:

Following the usual custom, I shall briefly relate the plan of the program of our Section, including the clinic, except that on small animals, or rather that of the Section on Small Animal Practice, for our clinic, as will be noted, does not confine itself to large animals; that is, the Section on General Practice still includes work with poultry, sheep and swine, as well as cattle and horses.

We have tried to include all these animals in our papers and demonstrations, although we have given special attention to cattle practice, which seemed to be the most important in this territory. While we have been able to secure practitioners to help somewhat in the program and clinic, it has been very difficult to get them, especially from a distance. However, the contributors who are not practitioners will furnish papers and demonstrations which should be highly interesting and instructive to men in the field of practice.

While our program deals exclusively with problems of diseases and treatments, both medical and surgical, still I have felt that some remarks along the line of the relation between the Association as a whole and the Section on General Practice, dealing with the question of making the A. V. M. A. as helpful for the practitioners as possible, may be beneficial at this time.

First, there has been much said and some attempts made to reorganize the Association so that practitioners would have more representation in it. Approximately 70 per cent of the membership are practitioners, yet, as a class, they have little to do with the workings of the Association. This is due to several factors, one of the chief ones being that a different group of practitioners usually attends the meetings each year, due to the fact that most of them must pay their own expenses to the meetings and lose their time and business besides when gone; so, generally speaking, any organized work in their behalf must be carried on with the support of the few who attend meetings regularly. With the other groups, or "blocks," many attend the meetings regularly, for they have their expenses paid and their salary goes on as well. This probably explains why some of the problems of practitioners have been neglected by the A. V. M. A. in the past.

Various efforts have been made to make the Association more representative of practitioners, such as the reorganization of the Association into a delegate system and later the increasing of the number on the Executive Board. In my opinion, such a drastic change as that recommending the delegate system is impracticable, for there are not the subordinate organizations to finance the movement. Besides, it is not desirable, for each member attending the meetings should have the right to vote and express himself upon any subject presented. Nor does the number attending the meetings prohibit the efficient conduct of the routine business.

As for the increase in the number forming the Executive Board, it is my observation that a body of the size we now have is more efficient than a large committee, which is hard to get together and hard to get to function also. What then is the way to make the Association more representative? I suggest that state associations be invited to send representatives to the meeting,

having the same vote and privileges as others in attendance. Another way is to allow expenses of practitioners serving on important committees, such as the Executive Board, and section officers, who spend a great deal of time getting up the program, so they can afford to accept these positions.

The problems of practice are fairly well covered in the report of the Policy Committee, which will be discussed at a general session. If this policy is carried out, it should do much to help iron out some of our difficulties. The Association is to be congratulated upon its adoption.

There are many ways in which practitioners themselves can improve conditions and assist in building up practice. One of the best advertisements of the profession is the individual practitioner who gives the community its first impression of the merits or shortcomings it possesses. Along this line of building up the individual efficiency of each practitioner, the Iowa Veterinary Association has recently secured from the Extension Department of the Iowa State College an agreement to furnish funds to send veterinarians from the Veterinary Division to hold one- or two-day conferences on groups of diseases or surgical conditions in any county asking for this aid—extension work for the farmers through the medium of the practitioners—a step which we consider will benefit the live stock owners much more than teaching a few of them to treat their own animals.

In solving the problems of practice, I have found the A. V. M. A. always willing to accept any reasonable, constructive suggestions that practitioners will make. It is up to us to present our problems in a fair light.

In conclusion, I wish to thank those who have contributed to the program, both by papers and demonstrations at the clinic. In our preparation of the program we have tried to follow the wishes of the local veterinarians who have offered suggestions, for we have thought that this is primarily their meeting and have appreciated their cooperation.

DR. B. W. CONRAD: Mr. Chairman, I move that this address, with the recommendations therein, be referred to the Executive Board. (The motion was seconded.)

CHAIRMAN STEEL: It has been moved and seconded that the address of the Chairman be referred to the Executive Board.

. . . The motion was carried. . . .

CHAIRMAN STEEL: Dr. L. H. Wright, secretary of the Section on General Practice, wrote me that he would not be able to attend this meeting, so I included in my paper something about the preparation of the program. Since Dr. Wright is not here, President Merillat has appointed Dr. J. N. Frost, of Cornell University, to act as secretary *pro tem.* We would like to have a few remarks from Dr. Frost.

DR. J. N. FROST: Having just been appointed to this position, I have no report to make, of course. Dr. Steel has worked out the program and I think that, if you will glance over it, you will find something of interest to all of you. I hope that we can get these sessions started on time, so that we may be able to get through with all the papers.

The following program was presented:

"The Effect on Live Stock of Water Contaminated with Fresh Water Algae," by Dr. W. Graham Gillam. (Published in the JOURNAL, September, 1925.)

"Possibilities and Limitations of Ovary and Testicle Transplantation in Veterinary Practice," by Dr. C. M. Haring. (Published in the JOURNAL, January, 1926.)

"The Influence of Diet in Hog Cholera Immunization Breaks," by Dr. Robert Jay. (In the absence of the author, this paper was read by Dr. David F. Fox, Sacramento, Calif. Published in the JOURNAL, November, 1925.)

ADJOURNMENT

THURSDAY MORNING, July 23, 1925.

The meeting was called to order at 9:30 a. m., by Dr. E. R. Steel, chairman.

The following program was presented:

"Case Reports on the Use of Oestrual Hormone and Other Gland Extracts in the Treatment of Functional Sterility," by Drs. H. S. Murphey, G. W. McNutt, B. A. Zupp and W. A. Aitken. (In the absence of all of the authors, the paper was read by Secretary Frost. Published in the JOURNAL, January, 1926.)

"Fowl Pest," by Dr. E. L. Stubbs. (Published in the JOURNAL, February, 1926.)

"Enzootic Hepatic Cirrhosis of Horses (Walking Disease) in the Northwest," by Drs. J. W. Kalkus, H. A. Trippeer and J. R. Fuller. (Published in the JOURNAL, December, 1925.)

"Laparotomy in the Cow," by Dr. J. N. Frost. (Published in the JOURNAL, January, 1926.)

CHAIRMAN STEEL: This concludes the literary part of the program. The next order of business is the election of officers. There are two officers to be elected in this Section; the Chairman and the Secretary. We will now entertain nominations for the office of Chairman.

DR. P. G. MACKINTOSH: I would like to make a motion that we retain the present Chairman, Dr. Steel, of Grundy Center, Iowa, for the ensuing year.

. . . The motion was seconded by Dr. J. R. Fuller. . .

. . . On motion duly made and seconded, it was voted that the nominations be closed and the Secretary instructed to cast the unanimous ballot of the members for Dr. E. R. Steel as Chairman of the Section on General Practice for the ensuing year. . . .

CHAIRMAN STEEL: The next is the office of Secretary.

DR. R. R. DYKSTRA: In view of the fact that the next meeting of the A. V. M. A. will be held in Lexington, Ky., which may properly be classed as being in the eastern section of the country, and a large number of the papers will in all probability be derived from that section, I think it is very important that the Secretary be an eastern man. Therefore, I would like to place in nomination the name of Dr. J. N. Frost for Secretary.

. . . The nomination was seconded by Dr. W. H. Lytle.

. . . On motion duly made and seconded it was voted that the nominations be closed and that the Chairman be instructed to cast the unanimous ballot of the members for Dr. J. N. Frost as Secretary.

. . . The meeting adjourned at 12:00 m.

ADJOURNMENT.

SECTION ON SANITARY SCIENCE AND POLICE

WEDNESDAY MORNING, July 22, 1925

The first session of the Section on Sanitary Science and Police was called to order by Dr. J. B. Still, of Winnipeg, Man., acting chairman in the absence of Dr. Orlan Hall, of Ottawa, Ont. In the absence of Dr. J. H. McNeil, of Trenton, N. J., secretary of the Section, Dr. F. J. Muecke, of Indianapolis, Ind., acted as secretary *pro tem*, these appointments having been made by President Merillat.

The following program was presented:

"Tuberculin Testing on the Western Range," by Dr. S. O'Toole. (Published in the JOURNAL, November, 1925.)

"Tuberculosis Eradication in the Northwest," by Dr. Samuel B. Foster. (Published in the JOURNAL, November, 1925.)

"County Herd Inspection Work in Oregon," by Dr. W. H. Lytle. (Published in the JOURNAL, November, 1925.)

"Teat Lesions in Cows Reacting to the Tuberculin Test," by Dr. Hadleigh Marsh. (Published in the JOURNAL, November, 1925.)

ADJOURNMENT.

THURSDAY MORNING, July 23, 1925.

The second session of the Section was called to order by the acting chairman, Dr. J. B. Still.

The following program was presented:

"State Meat Inspection," by Dr. J. P. Iverson. (Published in the JOURNAL, November, 1925.)

"Tick Paralysis," by Dr. E. A. Bruce. (Published in the JOURNAL, November, 1925.)

"New Phases in the Control of Anthrax Through Vaccination," by Dr. A. Eichhorn. (Published in the JOURNAL, December, 1925.)

"Bovine Abortion—Prevention and Control—Further Remarks," by Dr. M. F. Barnes. (In the absence of the author, this paper was read by Dr. E. L. Stubbs, of the Pennsylvania Bureau of Animal Industry, Harrisburg, Pa. Published in the JOURNAL, January, 1926.)

* * *

CHAIRMAN STILL: The next order of business is the election of a chairman and a secretary for the coming year.

MEMBER: I would like to nominate, as chairman, Dr. Edward A. Cahill, of Indianapolis, Ind.

DR. P. H. BROWNING: I would like to second the nomination of Dr. Cahill.

CHAIRMAN STILL: All those in favor with that nomination please stand up. (All stand.)

. . . Dr. Cahill is unanimously elected chairman. . . .

Now gentlemen, we are about to nominate someone for secretary.

DR. W. F. CREWE: I nominate Dr. N. F. Williams, of Texas, for secretary.

DR. C. H. SEAGRAVES: I second that nomination.

There being no further nominations, Dr. Williams was duly declared unanimously elected secretary for the coming year.

On motion the meeting was adjounred sine die.

ADJOURNMENT.

SECTION ON EDUCATION AND RESEARCH

WEDNESDAY MORNING, July 22, 1925.

The first session of the Section on Education and Research was called to order by Dr. F. M. Hayes, chairman.

(Chairman Hayes read his address. Published in the JOURNAL, September, 1925.) (Applause)

CHAIRMAN HAYES: We will now have the Secretary's report.

SECRETARY PICKENS: Mr. Chairman and Gentlemen, we have a very long and interesting program and I do not want to take the time to give a report, but there are two or three things I want to say.

In the first place, I wish to thank the members of this Section for their cooperation and assistance and I want especially to

thank the contributors. In discussing the papers I think it would be well, if each member, as he rises, will give his name and address so that the reporter will get the names and addresses correctly.

The following program was presented:

"Practical Results of Attempts to Control Abortion Disease," by Drs. B. T. Simms and Fred W. Miller. (Published in the JOURNAL, January, 1926.)

"Recent Studies in Ictero-hemoglobinuria of Cattle," by Drs. L. R. Vawter and Edward Records. (Published in the JOURNAL, January, 1926.)

"A Comparison of the Causative Organisms of La Mancha, Manqueira, European and American Blackleg," by Dr. A. S. Schlingman. (Published in the JOURNAL, January, 1926.)

"Infectious Bronchitis of Fowls," by Dr. J. R. Beach. (Published in the JOURNAL, February, 1926.)

"The Significance of *Bacillus Bronchisepticus* in Canine Distemper," by Dr. J. B. Hardenbergh. (Published in the JOURNAL, December, 1925.)

The meeting adjourned at 12:15 p. m.

ADJOURNMENT.

THURSDAY MORNING, July 23, 1925.

The meeting was called to order by the Chairman, Dr. Hayes, at 10:00 a. m.

The following program was presented:

"The Veterinarian, Society and the College," by Dr. E. E. Wegner. (Published in the JOURNAL, December, 1925.)

"Relation of the Thoracic Limb to the Thorax in the Case of the Horse and Ox," by Dr. J. D. Grossman. (Published in the JOURNAL, December, 1925.)

"Arteriosclerosis in Domestic Animals," by Dr. S. A. Goldberg. (In the absence of the author, this paper was read by title. To be published in the JOURNAL.)

* * *

CHAIRMAN HAYES: This brings us to the election of officers and nominations are now in order for chairman of this Section.

DR. WARD GILTNER: I would like to nominate Dr. E. T. Hallman, a man who has been a very faithful attendant at our meetings, but who was unable to be at this one on account of teaching duties. He will certainly be able to attend the meeting

at Lexington next year. He is one who, I feel, is well qualified to assist Dr. Pickens in getting up the program.

DR. C. M. HARING: I will second that motion. I would also move that the Secretary be instructed to cast the unanimous ballot of the Section for Dr. Hallman as chairman.

. . . The motion was duly seconded and carried. . . .

CHAIRMAN HAYES: Nominations are now in order for secretary of this Section.

DR. J. G. HARDENBERGH: I would like to nominate the present holder of the office, Dr. Pickens.

DR. GILTNER: I second the nomination.

DR. HARING: I move that the nominations be closed and that the President cast the unanimous ballot of the Section in favor of Dr. Pickens as secretary.

. . . The motion was duly seconded and carried. . . .

Dr. Pickens was declared elected secretary unanimously for the ensuing year.

* * *

DR. GILTNER: I move you, Mr. Chairman, that we extend a vote of thanks to the officers of this Section for their untiring energy and efficiency in providing us with a most excellent program this year.

DR. HARING: I will second the motion and, as it might be embarrassing for the President to do so, I will presume to put the motion. All those in favor of this motion will signify by the usual sign.

. . . Motion carried. . . .

CHAIRMAN HAYES: The chairman of the Section appreciates this vote of confidence. We have tried to make the program interesting. I wish to thank Dr. Pickens for his assistance because without him we would not have functioned as we have.

The meeting adjourned sine die.

ADJOURNMENT.

SECTION ON SMALL ANIMAL PRACTICE

WEDNESDAY MORNING, July 22, 1925

The first session of the Section was called to order by Dr. E. A. Ehmer, chairman.

Dr. Ehmer delivered his address.

* * * * * * * * * * *

GENTLEMEN:

It has been relatively only a few years since you men first became known

as small animal specialists. During this time, more and more men have turned to this field, until now—and the attendance at this session bears me out—it is considered the most acceptable and progressive branch of veterinary medicine.

Small animal practice has grown rapidly. The main factor in this, it seems to me, is the fact that while the pet animal, in most cases, represents little or no investment, it soon becomes a member of the family, and thus the human element enters in.

In this field, even with the great progress that is being made, the surface has only been scratched. This is a day of specialization. At a convention last year, one speaker called attention to the alarming fact that veterinarians were splitting up into special lines, and soon the profession would be a divided group of units.

It is my opinion that if anything has acted as a brake on the profession it has been the lack of specialization. If we only look at the medical profession, we can see what great benefits the specialist has brought to humanity. In our work, the same condition exists.

The lack of modern literature at present is a very great handicap. This condition is due not to a lack of men capable of writing it, but to the great expense and difficulty in marketing a scientific treatise. If we, as a group, hope to endure and to continue this rapid progress, the literature must be progressive. At present, nearly everyone who anticipates entering this field is confronted with the fact that there are no textbooks available. This can be overcome, and it must be considered and cared for by this group of men here present today.

Our journals are doing a most highly commendable work in soliciting and publishing the articles which have appeared, but we are not keeping up our end, for we are not writing enough articles. I have been assured that all articles worthy of printing would be gladly received. This then, will be one salvation in a literary sense. Instead of having two or three case reports and an article or two, let us send in a dozen of the former and half that many of the latter, each month, to each reputable journal. It is my hope that this Section will take definite action and, if possible, appoint the proper committeemen to begin the necessary work of creating small animal literature without delay.

As chairman of this Section, I realize the great responsibility that is ours. This is a departure from the usual custom. We have been honored by the Association in the creation of this Section. Let us guard that honor. The entire Association is quietly watching to see what our conduct will be here. Our desire as section officers is to make this a meeting that will eclipse all former meetings and which will bring favorable comment from our fellow practitioners the world over.

With the help of this splendid group of men, I know that this can be done, and we will now proceed to do it.

CHAIRMAN EHMER: We will now have the report of the Secretary, Dr. J. C. Flynn.

GENTLEMEN:

This is, you might say, an embryo organization, and I have not prepared a written report to give you. I might review the history of our efforts to secure the creation of this Section and to gain recognition for the small animal practitioner.

I believe very much in advertising in an ethical way. I do not believe that any individual practitioner should advertise that he can do work better than his fellow practitioner, but I believe that every veterinarian should advertise the fact that the veterinary profession is gaining in its ability to render service to the public. Veterinarians should not hide their light under a bushel, but should let the public know that they can do many of the things that the general public does not believe possible to be done.

There is no branch of the profession that advertises the veterinarian better than small animal practice. Here you come in contact with the very best clients, and you are called upon to handle a situation that will, if you conduct yourself properly, reflect a great deal of credit upon the profession.

The first time a Small Animal Section was talked of, to my knowledge, was at a meeting of veterinarians in Kansas City, several years ago.

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At the meeting in Des Moines last year, the matter was brought up on the floor, and we were promised at that time that the matter would be considered by the Executive Board. At the meeting of the Board, in Chicago, last December, the question was again considered, and the Board voted to create a Section on Small Animal Practice. President Merillat then appointed Dr. Ehmer as chairman of this Section, and he appointed me as secretary. He asked us to go ahead and prepare a program for this meeting, which we have done.

I want to make a few suggestions to you men who are interested in small animal work. Understand that this is the first meeting of this Section, and we possibly may be a little bit over-enthusiastic at times, but we want you to act with enthusiasm yourselves, and we want you, not only at this meeting but at future meetings, to see that we have an excellent program. There is nothing that will reflect more credit upon the veterinary profession than to have a small animal section well represented and a program that will reflect credit upon our Association.

Now, then, I want to impress upon you the urgent necessity for cooperation. When the secretary writes you a letter and asks you to appear on the program, it is evident that he wants to have you on the program. Do not write back and tell him that you are not capable, or that you do not have the time, or anything of the kind, but help to make your Section and your profession more worthwhile. (Applause.)

The following program was then presented:

"Abdominal Surgery," by Dr. William M. Bell. (To be published in the JOURNAL.)

"Canine Dental Surgery," by Dr. C. A. White. (To be published in the JOURNAL.)

"Hospital Sanitation," by Dr. J. F. McKenna. (In the absence of the author, this paper was read by title, and will be published in the JOURNAL.)

"So-called Salmon Poisoning in Dogs," by Drs. C. R. Donham and B. T. Simms. (The paper was read by Dr. Donham and illustrated with lantern slides. Published in the JOURNAL, February, 1926.)

"The Silver Fox," by Dr. H. M. Beckman. (In the absence of the author, this paper was read by title, and will be published in the JOURNAL.)

"Practical Feeds and Feeding of the Canine and Feline," by Dr. H. W. Brown. (To be published in the JOURNAL.)

ADJOURNMENT

THURSDAY MORNING, July 23, 1925

The second session of the Section was called to order by the chairman, Dr. E. A. Ehmer.

CHAIRMAN EHMER: Yesterday we had to adjourn before discussing the paper read by Dr. Brown. I am sorry that Dr. Brown is not here just now, but we will go ahead with the discussion of his paper. (See discussion.)

The following program was then presented:

"Diseases Affecting the Central Nervous System of the Canine and Feline," by Dr. O. V. Brumley. (In the absence of the author, this paper was read by title, and is published in this issue of the JOURNAL, p. 686.)

"Different Kinds of Canine Distemper. Symptoms and Treatment," by Dr. G. W. Closson. (To be published in the JOURNAL.)

"Prophylactic Immunization of Dogs Against Rabies by a Single Injection of Dead-Virus Vaccine," by Dr. A. S. Schlingman. (Published in the JOURNAL, December, 1925.)

"Internal Parasites of the Canine and Feline," by Dr. F. D. Patterson, Jr. (To be published in the JOURNAL.)

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CHAIRMAN EHMER: I think this would be a very good time to elect officers of this Section for the coming year. If we do not do it now, we may run over our time, or be rushed at the last minute. I will appoint Drs. Fowler, Kron, and Sleeth as tellers. Nominations are now in order for chairman of this Section.

DR. F. R. WHIPPLE: We have all read, and probably some of us remember, the old saying: "Let your light shine before men," and so on. This Small Animal Section owes a greater debt of gratitude to Dr. J. C. Flynn than to any other man in America. He is responsible for the establishment of this Section. For many years, he had an uphill fight, but he never tired working for the interests and for the establishment of this Section, and it is with a great deal of pleasure, gentlemen, that I present the name of Dr. J. C. Flynn for chairman.

. . . The nomination was seconded. It was moved that the nominations be closed, and that the chairman of the Section be instructed to cast the unanimous ballot of the Section for the election of Dr. Flynn as chairman for the Section for the ensuing year. The motion was seconded, and Dr. Flynn declared elected . . .

CHAIRMAN EHMER: Nominations are now in order for the office of secretary.

DR. F. D. PATTERSON, JR.: I wish to present the name of Dr. H. W. Brown, of Portland, Oregon, for the office of secretary.

. . . The nomination was seconded. . . .

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. . . It was moved that the nominations be closed and the secretary instructed to cast the unanimous ballot of the

Section for Dr. Brown as secretary. The motion was duly seconded and carried. Dr. Brown was declared elected.

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CHAIRMAN EHMER: We will now proceed with the program.

The following papers were then presented:

"Parasitic and Non-parasitic Diseases of the Ears of the Canine and Feline," by Dr. F. R. Whipple. (To be published in the JOURNAL.)

"Castor Oil," by Dr. O. A. Longley. (To be published in the JOURNAL.)

"Hospital Arrangement and Management," by Dr. Horst Schreck. (To be published in the JOURNAL.)

. . . The two latter papers, on motion duly made and seconded, were read but not discussed, owing to lack of time. . .

ADJOURNMENT

DETROIT HAS AIR MAIL SERVICE

With the inauguration of air mail service to and from Detroit, February 15, 1926, we are now only three hours away from Cleveland, four hours from Chicago, eight hours from New York, and just a few hours more than one day from San Francisco. Members are invited to post their letters by air mail, when replies are desired in the shortest possible time. The attention of our advertisers particularly is directed to the advantages of the air mail service for transmitting copy and proofs of advertisements.

EXAMINING BOARDS TAKE NOTICE

It has recently been brought to the attention of the Secretary that at least one spurious veterinary diploma from Harvard University is in existence. This diploma was presented to one of our veterinary examining boards by a veterinarian as his credential for a license to practice. The diploma in question is so poorly executed that there should be no difficulty in distinguishing similar fakes from a genuine diploma of Harvard University. For example, the bogus diploma bears a date of 1906, whereas the Harvard University Veterinary School ceased to exist in 1901. There are other important features which would readily help to identify similar spurious diplomas, if there are any in existence. Secretaries of veterinary examining boards are invited to correspond with the Secretary of the A. V. M. A. for further information, if interested.

ASSOCIATION MEETINGS

SOUTHEASTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

The winter meeting of the Southeastern Michigan Veterinary Medical Association was held in Detroit, December 16, 1925. The members of the Association were the guests of Parke, Davis & Company. A sumptuous repast was served in the company's cafeteria at six o'clock. The roll-call showed forty-seven in attendance.

The members were then escorted to the Medical Research and Biological Laboratories, where a short business session was held, followed by the literary program.

At the conclusion of the business session, President Sparhawk introduced Dr. Ward Giltner, of Michigan State College. Dr. Giltner's address turned out to be a presentation speech in honor of Dr. Joseph Hawkins, to whom Dr. Giltner presented a beautiful reading lamp in behalf of the members of the Association. Dr. Hawkins is a graduate of the Ontario Veterinary College, class of 1871, and is at present the oldest living graduate of the institution. In addition to this, Dr. Hawkins is the sole surviving charter member of the Ontario Veterinary Medical Association, and is one of the two surviving charter members and oldest ex-president of the Michigan State Veterinary Medical Association.

Dr. Hawkins was taken completely by surprise, but was able to accept the gift with a few appropriate remarks, in which he briefly reviewed the early history of the veterinary profession in Michigan.

For the literary program, President Sparhawk turned the chair over to the Secretary, so that he might have an opportunity to introduce to the members those of his former associates who were to address the meeting. The first subject was "Rabies," by Dr. A. S. Schlingman. He briefly reviewed the history of antirabic vaccination, from the time of Pasteur down to date. Dr. Schlingman also gave the latest compiled figures of the Detroit Board of Health, relative to the efficiency of rabies vaccination. Of 32,000 licensed dogs in Detroit, about 12,000 had been vaccinated during the past year, and about 20,000 not vaccinated. Of 177 cases of rabies which developed in licensed

dogs, only six cases were found in vaccinated dogs, while 171 of these dogs had not been vaccinated. In other words, the incidence of rabies among unvaccinated dogs was about twenty-two times as great as among vaccinated dogs in the same territory. A very animated discussion followed, led by Dr. B. J. Killham, state veterinarian of Michigan, who reviewed what had been done by the State authorities in the way of quarantines and other measures undertaken to prevent the further spread of the disease.

"Disinfectants and Insecticides. Description and Special Uses. Methods of Testing" was the subject of an address by Mr. H. C. Hamilton, who briefly reviewed the various classes of disinfectants, starting with the coal-tar group and finishing up with alcohol. The coal-tar group was characterized as that group, members of which produce milky emulsions when mixed with water. Then came those which make clear solutions, as liquor cresolis compositus, the high coefficient group, the mercury compounds, the silver preparations, the hypochlorites, hydrogen peroxid, iodin and the alcohols. Mr. Hamilton presented his subject in a manner that was intensely interesting and highly instructive. He explained the meaning of "phenol coefficient," the term used to designate the disinfectant power of a certain product. He later gave a demonstration of the method in his laboratory.

"Physiological Standardization of Drugs" was presented by Mr. L. W. Rowe, who enumerated the different groups of drugs which are now being physiologically standardized. These include some of the glandular extracts, such as adrenalin and pituitrin, and some of the vegetable drugs, such as ergot, cannabis, digitalis, and aconite. Mr. Rowe dwelt upon the value of standardization, and announced the fact that the 1926 United States Pharmacopoeia would recognize the physiological standardization of a number of the drugs he had mentioned. Mr. Rowe also gave a demonstration in his laboratory, later in the evening.

"Tetrachlorethylene. A New Anthelmintic" was the second subject presented by Dr. A. S. Schlingman, who briefly reviewed the history of this new anthelmintic, brought out by Drs. Hall and Shillinger, of the U. S. Bureau of Animal Industry. Dr. Schlingman compared tetrachlorethylene with carbon tretachlorid, which is likely to be supplanted, to a certain extent at least, by the newer drug, which is a closely related chemical compound. Dr. Schlingman gave a synopsis of the results of his

experiments on horses, cattle, sheep, dogs, cats, and chickens, to test the anthelmintic efficiency of tetrachlorethylene. In his laboratory, Dr. Schlingman later demonstrated the comparative effects of the two drugs, as shown by postmortem examinations, on young pups that had been previously given large doses of the drugs under discussion.

A vote of thanks was extended to Parke, Davis & Company for the splendid dinner and the excellent literary program which had been given the members of the Association.

H. PRESTON HOSKINS, *Secretary.*

DELAWARE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Delaware Veterinary Medical Association was held at the University of Delaware, Newark, December 18, 1925. The meeting was called to order by President H. P. Eves, of Wilmington. Dr. Eves, in a brief address, welcomed the members to Newark, and emphasized the value and importance of friendliness and cooperation between members of the profession. The President's address was followed by a report of the Secretary. Dr. F. P. Ruhl, of Milford, chairman of the Executive Committee, recommended Dr. Harvey Fell, of Wilmington; Dr. Frank Hare, of Dover; and Dr. W. C. Reeder, of Lewes, for membership in the Association. These men were elected members of the Association. Following the election of new members, the Society adjourned to the University Commons, where the members were entertained at luncheon as the guests of the Secretary.

At 1:00 p. m., the members assembled in Wolf Hall and proceeded with the program. The first subject upon the program was "Tuberculin Testing." This subject was well handled by Dr. Porteus, of the B. A. I., and Dr. Levinson, of Middletown. Dr. W. G. Middleton, B. A. I., in charge of tuberculosis eradication in Delaware, presented an interesting report upon "The Progress of Tuberculosis Eradication in this State." The discussion of this subject brought out the fact that, in a number of instances, owners of accredited herds are not continuing to have their herds tested. A motion was made that a committee, appointed by the president of the Association, go before the State Board of Agriculture with the recommendation that steps be taken to encourage owners of accredited herds to have their herds regularly tested by accredited veterinarians. The motion carried.

Dr. W. H. Wright, of Elkton, Maryland, gave a very interesting talk on his experiences in Texas during the recent foot-and-mouth outbreak. Dr. Wright brought back with him a number of excellent photographs showing lesions of foot-and-mouth disease. These photographs were passed among the audience.

As Dr. M. L. Zurkow, of Dover, who was scheduled to discuss his experiences in handling outbreaks of fowl plague, was unable to attend the meeting. His place upon the program was filled by the Secretary. The subject presented, however, was not fowl plague, but a report of some investigational studies on migration of the ova in cattle, and the relation of the sex of the bovine fetus to uterine position during intrauterine life. In the sex studies, the speaker presented a summary of the data obtained from a study of 100 pregnant cows, all members of the same herd, in which the horn position of the fetus was noted at the second or third month of pregnancy, and a correlation established between the horn position of the fetus and the sex as revealed at the time of birth. In this study, it was found that 60 of the cows were pregnant in the right horn, and 40 were pregnant in the left horn. Of the right-horn pregnancies, 28 were bulls and 32 were heifers. Of the left horn, 22 were bulls and 18 were heifers.

A round-table discussion of hog cholera vaccination brought out some interesting facts. Several members complained of receiving virus which is handled through the State Board of Agriculture, in which the expiration date was near at hand.

President Eves called upon the new members for a few remarks, and they responded, each giving a brief interesting talk.

At the election of officers, the old officers were reelected for another year. They are: Dr. H. P. Eves, president; Dr. C. C. Palmer, secretary; Drs. F. P. Ruhl, H. B. McDowell, and A. S. Houchin, Executive Committee.

C. C. Palmer, *Secretary.*

CENTRAL CANADA VETERINARY ASSOCIATION

The twenty-second annual meeting of the Central Canada Veterinary Association was held in the committee room at Howick Hall on November 25, 1925. The meeting was called to order by the President, Dr. J. A. Bean.

Dr. A. J. G. Hood, Chief Food Inspector, representing the Quebec Association, and a number of visitors were present, to-

gether with a good representation of the members of the Association.

After the reading of the minutes of the previous annual meeting and the auditor's report, showing the Association in good financial condition, applications for membership were received, and the following veterinarians elected to membership: Drs. K. L. Douglas, C. W. McIntosh, A. E. Cameron, W. R. Gunn, D. M. Robertson and J. E. Nesbitt.

The election of officers resulted as follows: Honorary patrons, Hon. W. R. Motherwell and Dr. J. H. Grisdale; honorary president, Dr. Geo. Hilton; president, Dr. J. A. Bean; vice-president, Dr. J. C. Reid; secretary-treasurer, Dr. W. H. Marriott. Council: Drs. O. Hall, F. Fisher, J. Bordeau, W. C. Young, Wm. Nicholls, J. B. Hollingsworth, P. W. O'Hara and Geo. Hilton.

A short report of the Committee on Contagious Abortion was presented by the Chairman, Dr. J. B. Hollingsworth.

Dr. C. M. Higginson, of Hawkesbury, presented a paper on "Diseases of Cattle from the Viewpoint of the Practitioner," which ably dealt with a number of diseases. Also a case report of actinomycosis, in a horse, requiring surgical treatment, and a number of cases of azoturia which responded satisfactorily to insulin treatment.

Dr. A. E. Cameron, Chief Veterinary Inspector, Ottawa, presented a paper entitled, "No-Visible-Lesion Cases in Tuberculin Reactors." This was very instructive and brought out a good discussion.

Dr. W. C. Young, of Almonte, Ont., presented a paper entitled, "Interesting Diseases of Cattle with Particular Reference to Contagious Abortion." This paper also brought out an animated discussion, in which Drs. Geo. Hilton, E. A. Watson, D. M. Robertson, W. R. Gunn and J. A. Bean participated.

"Poultry Diseases for which the Veterinary Practitioner is Called for Advice" was the subject of the paper by Dr. C. H. Weaver, of the Pathological Division, Ottawa. Dr. Weaver dealt with the most common diseases, including roup, chicken pox, intestinal parasites, white diarrhea and so-called deficiency diseases. He pointed out that the largest percentage of poultry losses was caused by parasitic infestations, and advised frequent and thorough disinfection of poultry houses and equipment, together with rotation of the runs and feeding-ground upon which the young chicks are placed after coming from the incu-

bator. Dr. Weaver pointed out that success lay in measures which would prevent disease, rather than in the medicinal treatment of infected birds.

Resolutions of condolence were passed in connection with the death of our Vice-President, Dr. A. W. Harris, and the tragic death of Mrs. W. C. McGuire.

W. H. MARRIOTT, *Secretary.*

KEYSTONE VETERINARY MEDICAL ASSOCIATION

On December 23, 1925, through the activity of Dr. C. J. Marshall, a meeting of the Keystone Veterinary Medical Association was called to order by President W. J. Crocker, at the University of Pennsylvania School of Veterinary Medicine. The Association has been dormant since May, 1918.

During the past few years the veterinarians of Philadelphia and vicinity have called their organization the Philadelphia Veterinary Club. The purpose of their meeting was to restore the old name. This was accomplished. It was voted that the officers elected October 9, 1917, should resume their duties and be continued in office.

By request, Dr. Alexander Glass gave an interesting talk on the early history of the Association and veterinary medicine in Philadelphia. He stated that Dr. William L. Zuill, Dr. W. Horace Hoskins, and himself, aged 24, 22, and 21 years respectively, located in this city of Philadelphia in the year 1882. At that time John Westley Gadsden, M. R. C. V. S., an Englishman, was the foremost veterinarian in the city. He was sober, industrious, gentlemanly and highly respected by all.

The three American graduates had much in common, and frequently met in the kitchen of Dr. Zuill's home to discuss veterinary subjects. The following year, Dr. E. A. A. Grange, of Toronto, Canada, came to Philadelphia, and he joined the other three in their conferences. Later Dr. W. B. E. Miller, Thomas B. Rogers and Charles T. Goentner were added to the list.

A code of ethics was drawn up and adopted by these men, and the Keystone Veterinary Medical Association was organized, October 7, 1882. There has been little or no change in the code of ethics since that time. The Association met at 1504 Sansom Street, in Dr. Grange's infirmary, and later in a room at 249 South 15th Street. Dr. Charles Michener was made an honorary member.

Mr. Fairman Rogers and Mr. Joshua B. Lippincott became interested in the profession and furnished the money for the purchase of ground and the erection of buildings to house the old University of Pennsylvania School of Veterinary Medicine.

Dr. Rush S. Huidekoper, a physician and man of social prominence in Philadelphia, was induced to go to Alfort, France, to study veterinary medicine. He became the first dean of the School.

Dr. W. Horace Hoskins led in legislative activities to protect the profession.

Dr. John Reichel spoke of the history of the Association since he had become a member, in 1906. He also spoke of the plan to organize a group to study canine distemper in cooperation with the proposed University of Pennsylvania Graduate School of Veterinary Medicine.

It was voted that a committee be appointed to review the old Constitution and By-Laws and recommend any changes that might be considered wise to meet the present needs. President Crocker appointed to serve on this committee Drs. John Reichel and C. J. Marshall.

G. A. Dick, *Secretary.*

NEVADA STATE VETERINARY ASSOCIATION

The annual meeting of the Nevada State Veterinary Association was called to order by President Given, in the Mining Building, University of Nevada, Reno, January 4, 1926. Roll-call showed eleven members and five visitors present.

The morning session was devoted to a business meeting, during which several amendments to the Association's constitution and by-laws were finally adopted, and preliminary arrangements made to send a representative of the Association to the conference of such delegates to be held at Lexington, Ky., August 16, 1926.

Dr. E. S. Glace, of Reno, was elected president for the ensuing year. Dr. W. C. Dye, of the U. S. Bureau of Animal Industry, was elected vice-president. Dr. Edward Records was re-elected to the office of secretary-treasurer.

The members and visitors of the Association were the guests of the Reno Rotary Club for luncheon, where Rotarian R. R. Dykstra, of Manhattan, Kansas, and Dr. J. A. Kiernan, of Washington, D. C., were the speakers of the day.

The literary program was given during the afternoon. Dr.

R. R. Dykstra gave a talk illustrated with charts on "Recent Surgical Developments." Dr. J. A. Kiernan spoke on "The Present Status of Cooperative Tuberculosis Eradication Work." Both of these interesting talks were discussed by those present, the speakers answering numerous questions in connection with the subjects presented by them.

The annual Association dinner was held at the Hotel Golden, during which Dr. Dykstra spoke very briefly but optimistically on the present status of the profession. Professor Peter Frandsen, of the Department of Biology, University of Nevada, gave a very interesting talk on the creation of the Department of Veterinary Science, at the University of Nevada, and its development up to the year 1905.

Final adjournment was taken at 8:30 p. m., to allow Drs. Dykstra and Kiernan and several members of the Association to take the train for Davis, California, to participate in the 1926 California Veterinary Conference, convening at that point the following day.

EDWARD RECORDS, *Secretary.*

KANSAS VETERINARY MEDICAL ASSOCIATION

The twenty-second annual meeting of the Kansas Veterinary Medical Association was held in Topeka, January 6-7, 1926. Fortunately the weather was favorable and a large attendance was present.

The meeting was called to order by President I. J. Pierson. The address of welcome was given by Major Ralph O'Neil, city attorney of Topeka, and State Commander of the American Legion.

Dr. S. L. Stewart responded to the address of welcome. Dr. I. J. Pierson gave a very interesting paper for the annual address as President. He was very optimistic relative to the future of the veterinary profession. His address was thoroughly enjoyed by everyone present.

Dr. C. H. Lerrigo, secretary of the Kansas State Tuberculosis Association, gave a very interesting and instructive discussion of the eradication of tuberculosis of humans. Dr. Lerrigo fully assured the veterinary profession that the physicians, and particularly the State Board of Health, were greatly interested in the tuberculosis eradication program, as being executed by the veterinarians of our State.

Dr. C. W. Bower gave a paper on the subject of "The X-Ray and Fluoroscope as an Asset to the Small Animal Practitioner." This paper was discussed by Dr. E. L. Quitman, of Chicago.

Dr. P. B. Darlington prepared and delivered a most excellent paper on "Anaplasmosis in Cattle." Dr. Darlington has been having some real experience with this disease in his practice and, of course, his paper was very interesting and instructive to all present. We hope Dr. Darlington will give us much more information concerning anaplasmosis in the future. His paper was discussed by Drs. S. L. Stewart, J. F. A. Thomas, Frank Breed and E. L. Quitman.

Beginning at one-thirty in the afternoon, Dr. S. L. Stewart, of Olathe, gave a very able paper, "Sterility in Cows—Observations, Prevention and Elimination." This paper was carefully prepared and well delivered. Dr. Albert Wempe gave a very good paper on "Uterine Diseases—Causes and Treatment in Cows." Dr. Wempe deserves much credit in preparing this paper. It was very instructive and thoroughly enjoyed.

Dr. Frank Breed, of Kansas City, talked concerning "Benefits Derived from the Use of Vaccines and Bacterins in Sterility, Ovarian, Uterine and Mammary Diseases." This brought out much discussion, which everyone enjoyed.

Dr. E. L. Quitman talked on "Small Animal Practice." This part of the afternoon program was thoroughly enjoyed, and was very instrucitve. A banquet was served in the evening. This was one part of the program that was surely a success. The writer has sat through many banquets of associations, both national and state, but never before has he enjoyed the whole evening as at this one. Everyone present really was disappointed when the last speaker had concluded. The time did not drag, as is so often the case at banquets.

Among the speakers for the evening were: Governor Benj. S. Paulen, of Kansas; Clyde Miller, secretary to the Governor; Dr. J. H. Burt, Dr. E. F. Kubin and Dr. White, of the Bureau of Animal Industry.

Thursday at nine o'clock we listened to a very interesting paper by Dr. A. T. Kinsley. Dr. E. J. Frick read a paper prepared by Dr. R. R. Dykstra, entitled, "The Future of the Veterinary Colleges and Their Graduates." We were very sorry that Dr. Dykstra was unable to be in attendance, but were pleased to have his paper.

A short business session was held and the following officers were elected: President, Dr. P. B. Darlington, of Chanute; vice-president, Dr. J. O. Young, of Topeka; secretary-treasurer, Dr. Chas. W. Bower, of Topeka (re-elected). Member of Executive Board, Dr. Sam E. Hays (re-elected). The Association voted to meet in Topeka again in 1927. In the afternoon a varied clinic was held consisting of surgical operations upon equine subjects, cows, goats, and the dog. Those participating in the clinic were: Drs. A. H. Gish, S. L. Stewart, F. R. Allerton, H. S. Richards, C. H. Gaines, H. C. Gale and E. L. Quitman.

CHAS. W. BOWER, *Secretary.*

CONESTOGA VETERINARY CLUB

The regular monthly meeting of the Conestoga Veterinary Club was held at the hospital of Dr. Harry W. Barnard, Lancaster, Pa., January 14, 1926.

Dr. H. W. Barnard presided. The roll-call showed fourteen members present.

Dr. W. J. Lentz, professor of anatomy and in charge of the small animal clinic at the Veterinary School of the University of Pennsylvania, held an interesting clinic and addressed the meeting on "Diseases of Small Animals." This lecture was the fifth of the present series being given by the University of Pennsylvania Veterinary Extension School.

HENRY S. WEBER, *Secretary.*

INDIANA-ILLINOIS VETERINARY MEDICAL ASSOCIATION

The Indiana-Illinois Veterinary Medical Association met at Vincennes, Indiana, January 29, 1926. Dr. H. H. Yocum, of Freelandville, Ind., vice-president, occupied the chair.

After the routine business was disposed of, Dr. Yocum presented a paper, "Publicity for the Veterinarian." This was followed by an interesting discussion.

Mr. Will L. TeWalt, secretary of the American Milk Goat Record Association and editor of the *Goat World*, gave an interesting address, which was thoroughly enjoyed by the veterinarians present.

M. W. SCOTT, *Secretary.*

NECROLOGY

V. N. ONISSIMENKO

Dr. Vassily Nicholaevich Onissimenko died of anthrax, at the Puunene Hospital, Maui, territory of Hawaii, January 13, 1926, in his 58th year.

A native of Russia, Dr. Onissimenko was a graduate of the Urieff Veterinary Institute, of Dorpat, Russia, in 1893. At the time of his death, Dr. Onissimenko was Deputy Territorial Veterinarian and Bacteriologist for the island of Maui, having held this position for about three years. Prior to his coming to Hawaii, he was Chief Veterinary Surgeon and Bacteriologist for the Chinese Eastern Railway, of Manchuria, 1912-1922. For five years, he was manager of a veterinary serological and bacteriological laboratory at Harbin. For nine months, he was veterinarian of a cavalry regiment in Siberia, at the time of the Boxer uprising, in China, about twenty-five years ago.

Dr. Onissimenko was the author of many articles on the subjects of rinderpest and hog cholera, published in Russian veterinary journals. In 1924, he presented a paper, in English, entitled, "Food Producing Animals of North Asia," before the Pan-Pacific Food Conservation Conference, held at Honolulu. He was a member of the Hawaiian Veterinary Medical Association, a member and former president of the Veterinary Medical Association of Manchuria, and had completed his application for membership in the American Veterinary Medical Association, January 1, 1926.

It is believed that Dr. Onissimenko contracted anthrax in his laboratory, as he had been working with some anthrax cultures, recently obtained from a Hawaiian plantation on one of the neighboring islands. He had a small pimple in back of one of his ears, and the belief was expressed that his fingers became infected and, in putting on or taking off his glasses, some of the infection was transferred to the pimple. The Honolulu *Star-Bulletin* contained a splendid editorial tribute several days following Dr. Onissimenko's death. He was characterized as a martyr to science, and was placed in the same class with Dr. Jesse W. Lazear, who died a martyr, some years ago, from yellow fever, which he contracted while investigating the disease.

JAMES COLE

"Dr." Jim Cole, 85 years old, of Winchester, Va., noted as a "cow doctor" of a generation ago, in Frederick and adjoining counties, died November 30, 1925. Although a non-graduate, he had a very extensive practice in Northern Virginia. Sick cows were his specialty. A sharp pocket knife constituted his instrument equipment. Linseed oil was his favorite therapeutic agent. He was a firm believer in the existence of such diseases as "wolf in the tail" and "hollow horn." "Dr." Cole was of imposing and picturesque appearance. He always carried a loaded rifle on his shoulder. Money was never a consideration with him. His wants were few; his fees were likewise light. He left no relatives. Friends agreed to defray the expenses of his funeral.

CHARLES T. BOYD

Dr. Chas. T. Boyd, of Harrow, Ont., died February 24, 1925. He was a graduate of the Ontario Veterinary College, class of 1915, and practiced at Harrow since his graduation. Death was due to heart trouble.

J. T. CROSBY

Dr. J. T. Crosby, of Kansas City, Mo., died November 8, 1925, after a brief illness. He was registered in Missouri as a graduate of the Royal College of Veterinary Surgeons (1884). He had been in practice in Kansas City for about fifteen years.

R. C. ERB

Dr. R. C. Erb, of Winnimac, Ind., was instantly killed, November 19, 1925, when he drove his automobile into a team of horses on one of the streets of his town. Dr. Erb was 39 years of age and had been a resident of Winnimac for about five years. He served in the Medical Corps during the War, and was ten months overseas. He is survived by his widow, his father, a brother and a sister.

WILLIAM T. RYAN

Dr. William T. Ryan, of Smyrna, Delaware, a licensed non-graduate practitioner, died November 24, 1925.

W. J. STERLING

Dr. W. J. Sterling, of Waterloo, Ont., died in St. Mary's Hospital, Kitchener, Ont., January 21, 1926, from pneumonia. He was sixty-two years of age.

Dr. Sterling was graduated from Ontario Veterinary College in 1881, and had been a resident of Waterloo for the past forty years. He held the position of Dairy Inspector of Waterloo, and took an extensive part in the sanitary work of the city. He is survived by five brothers and one sister.

ALBERT W. AUSTIN

Dr. Albert W. Austin, of Westville, Okla., died January 25, 1926. Death was due to concussion of the brain, following a fall on the ice.

Born at Breckenridge, Mo., August 26, 1870, Dr. Austin received his veterinary education at the Kansas City Veterinary College, from which he was graduated with the class of 1910. He served in the Spanish-American War, in Company K, Missouri Volunteers. He was a member of the Westville Lodge No. 186, A. F. and A. M.

Dr. Austin is survived by his widow, his mother, one sister, one daughter and one son. Interment was at Gravette, Ark., with Masonic ceremonies.

J. L. ADAMS

Dr. J. L. Adams, of Lake City, Florida, was killed in a shooting fray on the main street of the city, November 14, 1925. He was a non-graduate practitioner.

RICHARD Y. DAVISON

Dr. Richard Y. Davison, of Morton, Pa., died January 4, 1926. He was a graduate of the University of Pennsylvania School of Veterinary Medicine, class of 1900.

MRS. J. C. ELVIAGE

Mrs. J. C. Elviage, wife of Dr. J. C. Elviage (Ont. '90), of Erie, Pa., passed away very suddenly, in Tampa, Florida, January 24, 1926. Death was due to apoplexy.

BIRTHS

To Dr. and Mrs. W. J. Gray, of Thibodaux, La., a son, Walter Jasper, Jr., November 29, 1925.

To Dr. and Mrs. James S. Allen, of Nampa, Idaho, a son, Wendell Benson, December 9, 1925.

To Dr. and Mrs. Raymond H. Aull, of Dayton, Ohio, a son, John Forest, December 19, 1295.

To Dr. and Mrs. John Canty, of St. Johnsbury, Vt., a son, Dec. 30, 1925.

To Dr. and Mrs. C. A. Fridirici, of Fogelsville, Pa., a son, Curtis Alfred, January 18, 1926.

To Dr. and Mrs. Zac T. Nalls, of Jasper, Fla., a daughter, Alice Cyntha, February 8, 1926.

To Dr. and Mrs. A. A. Leibold, of Chicago, a son, February 11, 1926.

To Dr. and Mrs. Sivert Eriksen, of Mountain Grove, Mo., a daughter, Janet, February 15, 1926.

PERSONALS

Dr. J. W. Berry (A. P. I. '23) has located at Pulaski, Tenn.

Dr. George Schwamm (Corn. '21) is now located in Rochester, N. Y.

Dr. Daniel R. Kohler (Ont. '93) is president of the National Bank of Boyertown, Pa.

Dr. F. A. Imler (O. S. U. '06) is vice-president of the Kansas City U. S. D. A. Club.

Dr. John L. Tyler (Chi. '89) moved into his new home at Baldwin Park, Calif., March first.

Dr. N. O. Bevins (Iowa '11), formerly of Coon Rapids, Iowa, is now located at Hawkeye, Iowa.

Dr. F. G. Roth (Ind. '17), of Crown Point, Ind., has been reappointed County Veterinarian.

Dr. W. H. Emig (Ont. '07) has returned to Chattanooga, Tenn., to engage in the real estate business.

Dr. C. H. Wilmot (T. H. '17), of Chickasha, Okla, is reported to have been on the sick list for some time.

Dr. F. W. Crawford (K. S. A. C. '23) has requested a change of address from Colorado Springs, Colo., to Quincy, Ill.

Dr. James H. Harden (Corn. '23), of Brooklyn, N. Y., has taken over the practice of the late Dr. James Masterson.

Dr. H. D. Peterson (San. Fran. '18), of Dallas, Ore., is County Herd and Meat Inspector for Polk County, Oregon.

Dr. C. H. Atkinson (Wash. '20) has been transferred from Ripon to Modesto, Calif., with the State Dept. of Agriculture.

Dr. Hugo Hartnack (Giessen '13) has moved from Brooklyn, N. Y., to Chicago, Ill. Address: 20 East Jackson Blvd.

Dr. J. N. Shaw (Wash. '17), of Tillamook, Ore., has accepted a position as instructor at the Oregon Agricultural College.

Dr. H. A. Wilson (K. C. V. C. '13), State Veterinarian of Missouri, is an authority on snakes, both real and imaginary.

Dr. William T. Hume (Ind. '00), who has been located in Boonville, Ind., for twelve years, has removed to Evansville, Ind.

Dr. Geo. W. Clark (Wash. '12), formerly of Yakima, Wash., gives a new address—Sunnyside, Washington, R. F. D. No. 1.

Dr. G. H. Starr (McK. '17), of Norris City, Ill., has removed to Carmi, Ill., succeeding to the practice of Dr. McIntosh.

Dr. M. Donahoe (Chi. '17), who has been located at Breda, Iowa, has returned to his former location at Milford, Iowa.

Dr. J. R. Roche (Iowa '23), of Gowrie, Iowa, has entered the service of the U. S. B. A. I., and has been assigned to Chicago, Ill.

Dr. E. C. Goodrich (Gr. Rap. '15) has resumed practice in Kalamazoo, Mich., and limits his practice to dogs, cats, and foxes.

Dr. C. D. Williams (Chi. '07), after practicing at Woodbine, Iowa, for nineteen years, has decided to move to Red Oak, Iowa.

Dr. Louis Klotz (N. Y. U. '20), of Linden, N. J., has moved into more commodious quarters, at St. George Avenue and Adams Street.

Dr. John P. Stover (Ont. '93), of Greencastle, Pa., who has been seriously ill for some time past, is reported to be on the road to recovery.

Dr. A. C. Topmiller (McK. '07), of Murfreesboro, Tenn., was injured recently in an accident, on his way to attend a military funeral.

Dr. J. W. Roberts (San. Fran. '15), formerly of Turlock, Calif., is now associated with Dr. F. H. McNair (Corn. '05), of Berkeley, Calif.

Dr. C. G. Glendenning (Ont. '89), who was located at Clinton, Ill., retired from practice in June, and is now located at Fort Lauderdale, Fla.

Dr. W. S. Tucker (Ind. '02), of Waldron, Ind., has announced his candidacy for clerk of the Shelby County Circuit Court, on the Republican ticket.

Dr. A. R. Galbraith (Wash. '13) has disposed of his practice, at Garfield, Wash., to Dr. C. Vandermeer (Wash. '25), and moved to Centralia, Wash.

Dr. J. B. Hollenbeck (Ont. '95) has returned to his station at Bismarck, N. D., after four months spent in Texas on foot-and-mouth disease eradication.

Dr. V. J. Robinson (Chi. '09), of Atlantic, Iowa, has sold his practice and removed to Minneapolis, to engage in the furniture manufacturing business.

Dr. C. H. Schultz (U. P. '09), who has been in the Philippines for a number of years, is planning to return to the United States the latter part of March.

Dr. C. L. Winchester (Tex. '25) successfully passed the examination given by the Arkansas Veterinary Examining Board, in Little Rock, January 20, 1926.

Dr. H. Calvin Rea (K. C. V. C. '18) is the manager of "Cedarcroft," a hospital and boardir g kennels for dogs, located three miles out from Charlotte, N. C.

Dr. W. R. Spesard (McK. '14), of Greenup, Ill., has been appointed Shelby County (Ill.) veterinarian, to make a county survey of the bovine tuberculosis situation.

Dr. Thomas B. Carter (Ont. '14), of Portland, Ore., has been appointed County Herd Inspector for Tillamook County, with headquarters at Tillamook, Ore.

Dr. R. C. Julien (McK. '07), State Veterinarian of Indiana, broadcast from station WSBT (South Bend Tribune), January 27. His subject was "Tuberculosis in Cattle."

Dr. H. H. Seeley (Chi. '10), of White Hall, Ill., has been appointed Jersey County (Ill.) veterinarian, in connection with tuberculosis eradication work, with headquarters at Jerseyville.

Dr. J. A. Winkler (Cin. '18), of Newport, Ky., recently appeared before the city commissioners in behalf of an appropriation for tuberculin testing the cattle supplying milk to Newport.

Drs. S. R. Craver (Ont. '92) and Nevin S. Craver (O. S. U. '21), of 224 Holmes St., Youngstown, Ohio, have taken possession of their $35,000 hospital for small animals. This hospital is one of the best equipped in the State.

Dr. L. R. Haubrich (U. P. '12), of Claremont, N. H., lost his hospital and equipment in a fire of unknown origin, the night of February 11, 1925. The loss was partly covered by insurance. Several valuable animals were lost.

Dr. Walter H. Clawson (Cin. '13), of Hamilton, Ohio, received painful injuries recently, while cranking his car. The engine "kicked back," knocking Dr. Clawson's hand against the license plate, severing two tendons near the thumb of his right hand.

Dr. N. B. Smith (O. S. U. '93), of Billings, Mont., has been actively interested in getting a meat and milk inspection ordinance passed by the council of his city. The ordinance has been passed and the inspection work is to be under the supervision of a licensed veterinarian.

Dr. A. F. McGreevy (Gr. Rap. '15), of Sioux City, Iowa, is the owner of "McGreevy's King Pin," a three-year-old brindle bull terrier which has won thirty blue ribbons and eight special ribbons at various dog shows held throughout the United States and Canada.

Classroom Doggerel

"Yes," the teacher explained, "quite a number of plants and flowers have the prefix 'dog.' For instance, the dog-rose and dog-violet are well known. Can any of you name another?"

There was silence, then a happy look illuminated the face of a boy at the back of the class.

"Please, miss," he called out, proud of his knowledge, "collie-flowers!"—*The Progressive Grocer.*

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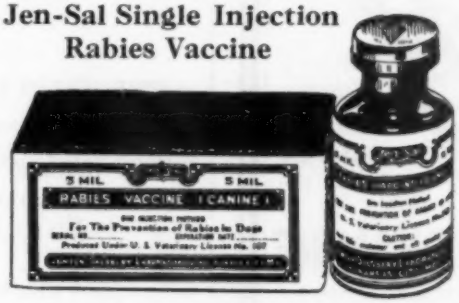

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*North American Veterinarian, August, 1925, page 38.

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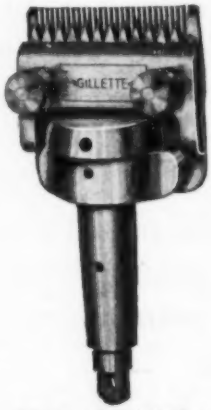

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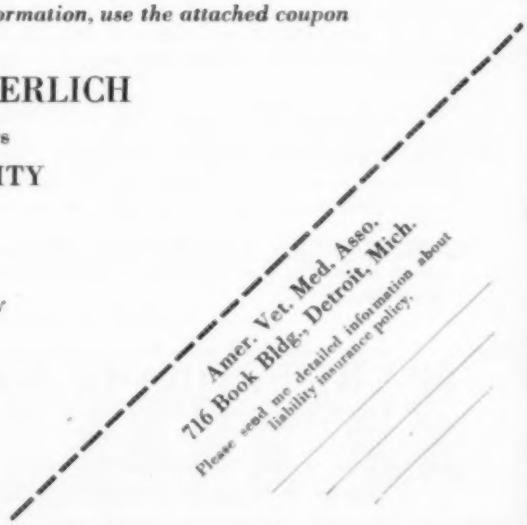
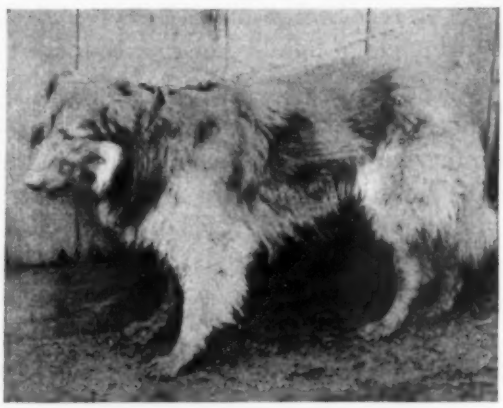


The Menace of RABIES



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